Manitoba Medical Review

Vol. 38 No. 8

October, 1958

Official Publication of the MANITOBA MEDICAL ASSOCIATION Winnipeg Canada

HEADACHE
FLASHES
HOT FLUSHES
DYSPNEA
PALPITATIONS
DIGESTIVE AND
URO-GENITAL ERETHISME

BELLERGAL

"SPACETABS"

Stabilize autonomic functions and relieve peripheral symptoms.

MAIN INDICATIONS: Menopausal disorders — premenstrual tension — sexual erethism — functional cardiovascular disorders — migraine prophylaxis.

EASY DOSAGE SCHEDULE: 1 Spacetab morning and night assures uninterrupted therapeutic protection.

SANDOZ PHARMACEUTICALS SANDOZ DORVAL, P.Q.

The Manitoba Medical Review

Vol. 38 OCTOBER, 1958 No. 8

Medicine

Observations on the Management of Acute Renal Failure

G. S. Varnam, M.D., F.R.C.P. (C)

Must has been written about the treatment of acute and chronic renal failure, but most of the recent literature outlines treatment schedules which require the use of special techniques which are not always readily or easily available to the physician who practices in a rural area with limited hospital and laboratory facilities. This paper will attempt to consolidate methods of treatment of acute renal failure, which can be carried out fairly easily in these areas with a minimum of investigative techniques. The use of extracorporeal dialysis, intestinal and peritoneal lavage will not be discussed since they are considered, and properly so, as measures to be instituted only under the control of specialists in this field.

Acute renal failure has been defined as that clinical syndrome resulting when renal excretory function is rapidly, but temporarily, lost because of alterations in renal circulation and the development of disseminated areas of renal tubular degeneration. In practice, it is considered that acute renal failure is present when there is sudden failure of the kidneys to produce at least 400 ml. of urine each day.

The causes of acute renal failure are many, but can be divided into those which are irreversible and those which are reversible when adequate treatment is instituted. Table 1, modified after Merrill (1), lists some of the causes of reversible acute renal failure.

Table 1

A. Following injury e.g. crush injuries, burns, etc.

B. Post-surgical shock, hemorrhage, etc.

C. Infections e.g. acute pyelonephritis, etc.

D. Circulatory insufficiency
   1. Water and electrolyte loss.
   2. Whole blood or plasma loss.
   3. Myocardial failure.
   4. Vascular failure—shock.

E. Nephrotoxins e.g. bichloride of mercury, bismuth, sulfonamides, etc.

F. Intravascular hemolysis e.g. incompatible blood transfusion, etc.

G. Hypersensitivity reactions.

H. Obstructive uropathy
   1. Intrarenal precipitation of sulfonamides.
   2. Intrarenal precipitation of uric acid crystals after therapy for leukemia.
   3. Obstruction of ureters e.g. tumor, etc.
   4. Ligation of ureters during surgery.

I. Acute exacerbation of chronic bilateral renal disease e.g. chronic glomerulonephritis, diabetic nephropathy, etc.

It should never be assumed by the attending physician that sudden cessation of urine production is due to acute renal failure. Other causes of oliguria should be sought immediately. A high urinary specific gravity and a high blood hematocrit make shock and dehydration suspect. An intravenous infusion of 500-1000ml. of 5% glucose in water may produce a sudden diuresis and confirm the suspicion that circulatory insufficiency is the cause of the oliguria. However, if a diuresis does not occur with one infusion, never under any circumstances try to force a diuresis by giving further infusions which will only produce fatalities.

If circulatory loading fails to reveal the cause of the oliguria, look for obstructive urinary tract lesions. These may be suspected in the presence of "anuria", defined as oliguria less than 100 ml. daily. If this search is unrewarding and the diagnosis of acute renal failure is sustained by the history, physical examination and laboratory investigation, treatment should be started immediately to enable the patient to survive the oliguric phase and to reduce morbidity.

Once the condition of acute renal failure has been established, it can be considered in two phases, the oliguric phase and the diuretic phase, as far as treatment is concerned. The oliguric phase is that phase during which urinary output remains below 400 ml. daily and the diuretic phase is that phase during which urinary output has risen above 400 ml. daily and continues to rise steadily, usually to a level of 2000 - 3000 ml, or more daily, with coincident loss from the body of those substances retained during the oliguric phase.

In the oliguric phase the commonest causes of death are overhydration and potassium intoxication. Hence, extreme caution must be exercised in the use of fluids and in the control of potassium levels in the body.

Management of fluid should be based upon estimation of insensible water loss from the skin and the lungs, endogenous water production and overt water loss due to urinary excretion, diarrhoea, vomiting and excessive sweating. Insensible water loss averages 1000 ml. daily and endogenous water production 500 ml. daily. Hence water replacement should be a maximum of 500 ml.

plus the replacement for overt losses daily allowing 100 - 500 ml. for each degree rise in body temperature (2). The best method to judge fluid requirements is to weigh the patient daily. In the absence of protein catabolism of significant degree the aim should be ½ - 1 pound of weight loss daily when oral feeding cannot be maintained. Before dehydration is clinically evident approximately 10% of body fluids are lost; it is safe to estimate a 10% increase in body fluids before frank evidence of overhydration is present. It should be remembered that small increments of excess fluid given daily soon amount to sizeable excesses of water in the body e.g. an excess of 50 ml. fluid daily amounts to 500 ml. excess in 10 days. Therefore, since the commonest error is to administer too much fluid, it is probably much wiser to restrict fluid and give less than the calculated daily requirement.

Control of body potassium in acute renal failure depends upon many factors. Normal catabolism of body proteins is accompanied by release of potassium and acid metabolites such as phosphates and sulphates. Stress, infection, trauma, surgery, devitalised tissue and the presence of hematomas accelerate protein catabolism and hence the release of potassium and acid metabolites. The early correction of these additional factors is essential in the control of potassium levels and the prevention of potassium intoxication. The intake of 100 gms. glucose daily halves protein catabolism and helps to remove phosphate and sulphate ions and so prevent acidosis and ketosis. Because most patients in acute renal failure do not tolerate oral feedings too well, this glucose should be given intravenously. Since fluid is restricted, these infusions should be 15-25% solutions injected through a high polythene venous catheter to minimize venous sclerosis. An attempt should be made to provide as many calories as can be given safely within the limits of fluid restriction. The use of 50% fructose solutions in addition to the hypertonic glucose infusions is very helpful in these situations.

Hyponatremia, acidosis, hypocalcemia aggravate hyperkalemic effects. The net result of the actions of these several factors may be followed easily by serial electrocardiograph tracings which show changes in conduction with high, peaked T waves, prolonged QRS complexes and eventual production of a sine wave complex as the condition progresses. The effects of hyperkalemia may be reduced for short periods of time by intravenous injections of 10% calcium gluconate or by the addition to the intravenous infusions of one unit of regular insulin for every 2 grams of glucose. The use of sodium salt infusions to counteract hyponatremia is not recommended because of the associated danger of death due to overhydration and pulmonary edema. Rather, further restriction of fluids should be carried out.

An infusion mixture (3) which has been found useful in the treatment of hyperkalemia to minimize its "toxic" effects is composed as follows:

Calcium gluconate 10% 100 ml.
$NaHCO_3$ ..... 7.5% 50 ml.
Dextrose 25% in water with 50 units regular insulin added—400 ml.
Isotonic NaCl or 1/6 M Na lactate—Volume of output.

Anemia frequently complicates acute renal failure. Blood transfusion is often necessary to correct this condition. However, it should be remembered that the erythrocytes release about 2.2 mEq/L/day of potassium in stored blood. To obviate this difficulty and to reduce the possibility of overhydration, only packed, washed erythrocyte transfusions should be given to keep the hematocrit at approximately 30%.

Cation exchange resins (4), in general, should not be used to attempt to reduce potassium levels in the body. Such resins have sodum or acid radical cations as exchange cations and these may be dangerous in themselves, sodium cations increasing the risks of overhydration and acid radical cations the risk of acidosis.

It should be remembered always that the best treatment for potassium intoxication is prophylaxis—do not give oral potassium, treat infections, drain blood accumulations, débride devitalised tissue, give adequate non-protein calories, stimulate intestinal losses with non-toxic cathartics and suction, if vomiting and diarrhoea are not present, and use glucose infusions with added insulin to promote potassium uptake during carbohydrate metabolism.

Medications which are excreted by the kidneys should be used with extreme caution or not at all. Digitalis should be given intravenously only with great care. Its administration should be followed by serial electrocardiograph tracings. In any event, never give a digitalizing dose at a single injection. Phenobarbital and barbital should never be used for sedation since they are excreted by the kidneys. Foods rich in potassium e.g. orange juice, must not be given in the oliguric phase. BAL, used in the treatment of heavy metal poisoning, is excreted to the extent of 80% of the ingested dose and must be used with caution.

In the diuretic phase the physician often relaxes his vigil with the feeling that this has been "a job well done". This may be a fatal relaxation, the patient often succumbing to such complications as pulmonary infection, pulmonary edema and pulmonary embolus.

Never try to force a diuresis in this phase and never try to replace water losses fully, provided hydration is adequate and sodium concentration is low. The diuresis is an attempt to correct overhydration which is the usual event in the oliguric phase.

As urinary output exceeds 1500 ml./day it is no longer necessary to restrict potassium intake and potassium may be given ad lib. By this time oral intake usually has been re-established and dict, including protein, may follow the whims of the patient. The diuresis which occurs frequently results in a rise in non-protein nitrogen because of the greater loss of water compared to that of non-protein nitrogen. This situation is not a cause for alarm and generally should be ignored provided fluid and electrolyte balance is otherwise satisfactory.

When damage to the central nervous system and to the mechanisms regulating the tonicity of body fluids occurs, there is preferential retention of sodium. A urinary PH less than 5 suggests this condition and serum sodium estimations confirm it. The treatment in this case is to restrict sodium intake.

Finally, it cannot be stressed too strongly that the treatment of any patient with acute renal failure must rest with a **single** physician to prevent fatalities due to duplication of orders. Acute renal failure cannot be said to be a disease from which the patient usually recovers in spite of the physician; rather it is a disease of which patients frequently die because of mismanagement.

The following case report illustrates how insidious in their onslaught overhydration and potassium intoxication can be in the absence of physical signs: H.R.J., age 32, was admitted to hospital with a history of chronic dudenal ulcer complicated by intractable pain uncontrolled on medical management. A subtotal gastrectomy was performed with uneventful post-operative course until the 3rd post-operative day. At this time the patient became nauseated with a tachycardia of 100. On the 5th day diarrhoea began. On the 7th day his pulse was 160, he was flushed, suffered from excessive sweating and continued to have diarrhoea. Urinary output started to decrease. By the 8th day oliguria was firmly established and a diagnosis of acute renal failure was made. The oliguric phase included the 7th to the 13th post-operative days. The diuretic phase began on the 14th day and lasted until death occurred on the 16th post-operative day. The laboratory findings for these periods are shown in Table 2.

From this table it can be seen that the oliguric phase must have preceded the 7th post-operative day because of the high BUN but this situation was not recognized at that time because detailed

Table 2

Post-operative Day	K m Eq/L	Na m Eq/L	Cl m Eq/L	BUN mg%	CO_2 Comb. Power Vol. %	WBC	Hematocrit %
8th	4.4	130	106	128		38000	50
9th	3.8	140	97	124	30		45
10th	4.8		101	144		30500	
11th	4.1	137	90	148			
12th	3.8		81	188	24		
13th	4.7	122	70	240			
14th	4.9	128	74	248		21000	37
15th	4.9	123	78.3	320			
16th			85	304	24		

intake and output control was not instituted until the eighth day. The patient was obviously dehydrated on the 8th day but by the 14th day the hematocrit and the serum sodium indicated overhydration.

Fluid management showed that, during the oliguric phase, the intake and output were 14,850 ml. and 7,160 ml. respectively and that, during the diuretic phase, were 10,125 ml. and 3,835 ml. respectively. Accounting for the insensible water loss, for the endogenous water production and for the water loss of approximately 900 ml. due to temperature elevation, the net excess of administered fluid over the 9 day period preceding death of the patient was 8,580 ml.

Even though the serum potassium was normal throughout the course of this illness, the electrocardiograph showed unequivocal evidence of hyperkalemia during the last 8 days of life. Hyponatremia and acidosis obviously played a large part in this combined effect.

Summary and Conclusions

1. The treatment of acute renal failure has been discussed from the standpoint of the rural physician with limited diagnostic and treatment facilities.
2. Some of the common errors in management have been emphasized.
3. A typical case report has been summarized to illustrate the salient features of the discussion.

References

1. J. P. Merrill: The Treatment of Renal Failure, Grune and Stratton, Inc., New York, P. 71, 1955.
2. Farber, S. J.: "Emergencies related to acute and chronic renal failure". Med. Cl. North America, 657, May 1957.
3. Meroney, W. H., and Harndon, R. F.: "The Management of acute renal insufficiency". J.A.M.A., 155: 877, 1954.
4. Strauss, M. B., and Raisz, L. G.: "Treatment of acute renal shutdown". Arch. Int. Med., 95: 846, 1955.

The Hereditary Hemolytic Anemias

Alvin Zipursky, M.D.

For many years it has been recognized that certain inherited diseases are characterized by a shortened red blood cell life span. These diseases are referred to as the Hereditary Hemolytic Anemias (Table 1). Recent progress in medical research has demonstrated that these diseases are actually "inborn errors of metabolism" of the erythrocyte. An attempt will be made to describe herein some of these defects in red cell metabolism and their relation to the clinical and pathological manifestations of the specific diseases.

Table 1

Hereditary Hemolytic Anemias

(Inborn Errors of Red Cell Metabolism)

1. Abnormalities of Shape and Structure
   a. Hereditary spherocytosis
   b. Hereditary elliptocytosis
   c. Congenital non-spherocytic hemolytic anemia
2. Abnormalities of Hemoglobin
   a. Altered hemoglobin structure
      (sickle cell anemia, hemoglobin C, D, E, G, H, I, J, K, L)
   b. Altered hemoglobin production (thalassemia)
3. Abnormal Sensitivity of Erythrocytes to Certain Drugs
   a. Primaquine sensitivity
   b. Favism
   c. Naphthalene sensitivity

The red blood cell was once thought of merely as an inert bag of hemoglobin enclosed within a semi-permeable membrane. It is now recognized that this cell is, on the contrary, an actively functioning unit utilizing energy for the performance of its many vital functions. It is therefore conceivable that abnormalities in the metabolism of the erythrocyte may result in a shortening of its life span.

Before discussing the hemolytic anemias it would be best to first consider a few of the more important features of red cell metabolism.

Erythrocyte Physiology

The erythrocyte is a cell consisting of about 63% water and 33% hemoglobin. The structure of the cell is made up primarily of lipoproteins (stroma) which are heavily condensed at the surface into a "membrane" but thin out into a meshwork deeper in the cell[1]. It is within this meshwork that hemoglobin and most of the energy-producing elements of the cell lie[2, 3].

The material lying within the stroma mesh performs a great number of functions. A living red cell consumes glucose, prevents methemoglobin accumulation, maintains high potassium levels and prevents breakdown of the cell and lysis[2]. If energy production is interfered with, methemoglobin will accumulate, potassium levels will fall and the cell will quickly lyse.

In the erythrocyte energy is obtained exclusively from the breakdown of glucose to lactic acid (glycolysis). As glucose breaks down it does so by passing through a series of phosphate compounds. The separate steps concerned in this pathway are not important herein; what is important is that energy is obtained from glucose by being handed along a series of phosphate ester "stepping stones," finally becoming available for the red cell's many functions.

Glucose may be broken down anaerobically (without oxygen) or aerobically (with oxygen). In the red blood cell the former mechanism is the major one but it appears that the latter also has an important role.

In the aerobic consumption of glucose, oxidation occurs and, with it, the formation of reduced triphosphopyridine nucleotide (TPNH)[2]. Certain functions within the red blood cell are dependent on a continued supply of TPNH (e.g. methemoglobin and glutathione reduction) and deficient production of TPNH will result in abnormalities of these functions. With these few concepts in mind we can now examine some of the pathological processes involved in the Hereditary Hemolytic Anemias, and observe how each of the above physiologic processes may be altered in disease states.

Sickle Cell Anemia

Sickle cell anemia is a disease characterized by a severe hemolytic anemia and repeated thrombotic episodes. The salient feature of the disease is that the red blood cells assume bizarre "sickle" shapes when exposed to lowered oxygen tension.

In 1949, Pauling et al[3] reported that the hemoglobin of patients with sickle cell disease migrated at a different rate in an electrophoretic field than normal hemoglobin. He postulated that this difference in mobility may be due to the "sickle" hemoglobin possessing fewer negatively charged particles than normal hemoglobin. Recently, this hypothesis appears to have been confirmed by the studies of Ingram[4] who has demonstrated that sickle hemoglobin possesses two valine molecules, where the normal hemoglobin has two glutamic acid radicals. This would then leave "sickle" hemoglobin with two less carboxyl groups and therefore two less negatively charged ions.

How then can this slight alteration in the hemoglobin molecule bring about such a profound change in red cell metabolism? The answer is not yet known but a partial picture can be drawn. Hemoglobin is a combination of a protein (globin) and a porphyrin (heme). The abnormal charges on the sickle globin molecule appear to favor intermolecular union; this can be prevented by maintaining heme in an oxygenated state. When heme is de-oxygenated, the globin molecules can approach one another so closely that intermolecular union occurs and gel-like structures are formed. The latter have a characteristic "sickle shape" as seen through the electron microscope, and are referred to as tactoids. It was mentioned earlier that hemoglobin lies in intimate relation with the stroma (ultrastructure) of the red blood cell; for this reason these changes in shape of the hemoglobin mass can cause a distortion in the cell stroma and membrane with assumption of the

characteristic sickle-shaped cell. When the cells are "sickled" the viscosity of the blood rises sharply[5], hence explaining the tendency to thrombosis. These bizarre alterations in red blood cell structure are not without effect on the metabolism of the cell; when sickling occurs sodium and potassium exchange is altered[6], phosphate entry is slowed[7], and the viability of the cell is diminished. Thus the close association between hemoglobin and stroma within the erythrocyte allows an abnormal hemoglobin to exert profound influence on the shape and function of the entire cell.

Hereditary Spherocytosis

Hereditary spherocytosis is a disease characterized by an abnormal shape and viability of the erythrocyte. These cells are small and spherical instead of bi-concave. The spherical shape results in an increased mechanical and osmotic fragility. Emerson et al[8] demonstrated that spherocytes are trapped within the normal spleen, much more readily than normal cells. They suggested that this selective trapping was due to the abnormal shape of these cells. Furthermore, the spherocyte, on standing with no nutrient, breaks down more readily than normal cells. Thus the hemolysis characteristic of this disease appears to be due to the splenic trapping of the spherocytes which are, in turn, destroyed by their inability to withstand the stagnation within the splenic pulp.

But what causes the abnormal shape or the inability to withstand stagnation? In 1955, Prankerd and Altman shed some light on this problem by their discovery that the glycolytic cycle of spherocytes differed markedly from that of normal cells. They found that radioactive phosphorus was incorporated very slowly into phosphate esters ("stepping stones of glycolysis") within spherocytic cells. Tabechian et al in 1956 suggested that the defect in hereditary spherocytosis may be that of a single enzyme in the glycolytic cycle.

If we accept that spherocytes have a defect in their glycolytic cycle, how can we correlate this with the observed abnormalities of shape and viability? The shape of the red blood cell is maintained by the metabolism of the cell. If metabolism ceases the cell will lose its biconcave shape and gradually become spherical. Furthermore, the erythrocytes of oxen are normally spherical and also have a low turnover of phosphate esters within the stroma[9]. These findings suggest that the abnormal shape of the spherocyte may be related to its abnormal metabolism. If erythrocytes are deprived of glucose, or if glycolysis is inhibited, the cells will rapidly break down on standing; this tendency is greatly increased in spherocytic cells with their abnormal glycolytic cycle. Cells trapped within the spleen are subject to stagnation and deprivation of substrate; the more susceptible spherocytes may therefore succumb sooner.

Thus, it is conceivable that an abnormality in the energy-producing system within the erythrocyte can result in the red blood cell shape and viability changes characteristic of the disease, Hereditary Spherocytosis.

Primaquine Sensitivity

The erythrocytes of 10% of American Negroes are very sensitive to certain drugs such as primaquine or phenylhydrazine. This is a hereditary defect of the erythrocyte and becomes manifest as a severe hemolytic anemia upon exposure to any of the specific drugs.

Beutler et al discovered that these cells had low levels of reduced glutathione. The latter is a tripeptide whose function in the erythrocyte is unknown. Furthermore, it was found by these authors that when phenylhydrazine was added to these cells in vitro the levels of reduced glutathione declined more rapidly than normal cells. These and other findings suggested that these cells had a lesser ability to reduce glutathione when compared to normal cells. Within the erythrocyte glutathione is reduced by a specific enzyme which requires TPNH as a co-enzyme. It will be recalled that TPNH levels in the red cell are dependent on normal aerobic glycolysis. Accordingly, Carson et al examined the aerobic cycle of glycolysis in these cells and found that there was a marked deficiency of one of the oxidative enzymes and hence a decreased production of TPNH. Thus it would appear that the specific defect in these cells is a lack of a single enzyme. How hemolysis with exposure to the specific drug actually occurs is unknown, but it appears to be related to this enzyme deficiency.

These three diseases have taught us that red cells can be profoundly affected by changes at a molecular level. An abnormal hemoglobin molecule, altered energy-producing system or the lack of a single enzyme can seriously affect the metabolism of the erythrocyte, producing the clinical picture of a hereditary hemolytic anemia. In the future we hope to be able to recognize such diseases not only as clinical and pathological entities but also by the nature of their fundamental molecular abnormality.

References

1. Ponder, E. Blood, 9: 227, 1954.
2. Prankerd, T. A. J. Brit. J. Hem., 1: 131, 1955.
3. Pauling, L., Itano, H. A., Singer, S. J., and Wells, I. C. Science, 110: 543, 1949.
4. Ingram, V. M. Nature, 180: 326, 1957.
5. Harris, J. W. Proc. Soc. Exp. Biol. & Med., 75: 197, 1950.
6. Tosteson, D. C., Carlsen, E., and Dunham, E. T. J. Gen. Phys., 39: 31, 1955.
7. Prankerd, T. A. J. Clin. Sc., 14: 381, 1955.
8. Emerson, C. P., Shen, S. C., and Castle, W. B. J. Clin. Invest., 25: 922, 1946.
9. Prankerd, T. A. J., Altman, K. I., and Young, L. E. J. Clin. Invest., 33: 957, 1954.
10. Tabechian, H., Altman, K. I., and Young, L. E.: Proc. Soc. Exper. Biol. & Med., 92: 712, 1956.
11. Beutler, E., Robson, M., and Buttenwieser, E. J. Clin. Invest., 36: 617, 1957.
12. Carson, P., Flanagan, C. L., Ickes, C. E., and Alving, A. S. Science, 124: 484, 1956.

Pathology

The Use of Cytology in Diagnostic Medicine

D. W. Penner, M.D.

The use of cytology in diagnostic medicine is not new. In hematology, examination of the peripheral blood cells, and at a later date the bone marrow has for a long time been a routine procedure. Dr. George Papanicolaou has done much in America to stimulate interest in the field of cytologic diagnosis. Yet nearly 100 years ago excellent cytologic description of cancer cells in ascitic and pleural effusions was published. In the last ten years much has been written about the detection of cancer by cytologic smears, especially cervical carcinoma. Few medical subjects have reeived wider publication in the lay papers and magazines. On October 11th, 1957, the following appeared in the Free Press in the Medical Memos column:

"If you are a woman over 35 have your doctor give you the Pap smear test.

Pap refers to Dr. Papanicolaou who developed a simple, quick, painless procedure which indicates the possibility of cancer of the uterus long before any symptoms appear or any sign of it can be seen by the doctor.

It means that practically no woman ever need die from this type of cancer which is quite common. If detected early enough, it is 100% curable.

The doctor merely aspirates a small amount of the secretion from the vagina with a glass syringe and smears this material on a glass slide. It is then examined by a technician who is specially trained to make these microscopic studies. If the cells in the secretion are suspicious of cancer it is still necessary to depend on biopsy for conclusive proof. Biopsy means the surgical removal of a small piece of the tissue for microscopic examination.

If cancer is found then the uterus is removed. Because this organ is out long before the cancer has a chance to spread to any other part of the body, your life has been saved.

The test should be done once a year regardless of how good you feel and even though you have no unusual symptoms."

Cytologic diagnosis apart from hematology may be applied to cells shed or scraped from the cervix, endometrium, G.I. tract, respiratory tract, various effusions, C.S.F., urine and to needle aspirated cells from any accessible tumor in the body. Both these techniques have a most useful place in modern medicine and should be more used where facilities are available for interpretation.

Presented at the Annual Convention of the Manitoba Medical Association, October, 1957.

In spite of the fact that cytologic techniques were introduced in the Winnipeg General Hospital Laboratory 14 years ago many physicians seem unaware that this interpretive service is readily available to any Manitoba physician. The volume of work in this specialty remains relatively small.

Last year 1,050 cytologic examinations were made. This is somewhat less than 1/10th of the number of biopsy and surgical specimens examined. Over half of these (551) were from the female genital tract, 226 were from the respiratory tract and 174 from pleural and ascitic effusions. The remainder included aspiration biopsies and material from the urinary tract, secretion from breast, gastric and oesophageal washings, etc.

Female Genital Tract

In our material 490 different cases were documented with a toal of 551 examinations. Sixty-one and 47 (8.4%) were positive for cancer. In over half of these positive cases (32 cases) cytology was the primary or initial method of diagnosis. Eight cases of 1.5% of the total cases examined, were in situ or non-invasive carcinoma. The ultimate aim in any cancer program is cancer prevention, but falling short of being able to achieve this, the next best thing is early diagnosis and treatment. If a diagnosis of carcinoma of the cervix can be made before the tumor invades, a cure can be assured even with very conservative surgery. An in situ or non-invasive carcinoma cannot be seen or felt and it produces no symptoms, although it is frequently associated with the signs and symptoms of a benign cervical erosion. Detection of this condition can best be done by cytologic examination and diagnosis confirmed by tissue biopsy. In all positive smears in the absence of a gross lesion, a cold knife conization of the cervix should be performed for evaluation of the extent of the cancer.

Selection of Patients

What patients should have smears taken: Ideally all women over the age of 24 should be screened. Many mass incidence studies are currently being conducted. The largest is under Dr. C. Erichson, a Memphis, Tennessee pathologist, who recently published his preliminary findings in which an incidence of cancer found was 8/1000. This included both invasive and non-invasive carcinoma. In situ carcinomas have an incidence of probably not more than 1-2 cases per 500 screened. (J.A.M.A. 162: 167, 1956).

Perhaps before too long it will be practical to do a screening of the female population in Manitoba. I do not know how practical it would be to do a smear on all new adult female patients seen in your practice, but certainly it would seem advisable to take more than is currently being done.

Cost

Where a charge is made, it is $3.50 in Manitoba, less than a routine blood count or chest x-ray.

Method of Obtaining Material from the Cervix

Most workers prefer material obtained directly from the squamo-columnar and endocervical area rather than an aspiration of vaginal secretion. This is easily obtained by scraping or cotton on an applicator technique. Material from the endocervical canal can be obtained by use of a small aspiration pipette. The material obtained should be smeared on a clean dry glass slide, air dried and submitted for examination.

Method and Availability of Interpretive Service:

Screening of material is usually done by a well trained and competent cytologic technician, with final interpretation by a pathologist. Up until recently both screening and final interpretation were done by the pathologist. In both Canada and the United States less than half of the laboratories have technician screeners. These people are in very short supply. In Manitoba, smears can be submitted through the rural diagnostic biopsy service or to the various pathologists trained in this field. In our laboratory we now have sufficient technical and professional personnel to handle ten times our present volume.

Accuracy

Cervical cytologic procedures should always be looked upon as detection or screening procedures rather than definitive diagnostic, and all cytologic interpretation should be confirmed by histologic tissue examination. Dr. Erichson and others have pointed out that in any screening procedure a balance must be achieved between sensitivity or incidence of detection and accuracy or reliability. The greater the accuracy the fewer will be the cases detected, whereas, if all abnormal smears, but not diagnostic of carcinoma are selected for further investigation by repeat smear and biopsy, a greater number of cancers will ultimately be discovered. A very small percent of false positives are inevitable since very abnormal cells may be found following infected abortions, in polyps and following the use of radiation. This is usually in the order of 1% plus.

The second most useful field for cervical cytology is in appraising and following the use of radiation for treatment of carcinoma of cervix. Work is now being done which suggests that it may be possible to predict which tumor will be radio-responsive and which will be radio-resistant. Repeat cervical smears taken during and after the use of radiation can be most useful.

Pulmonary Cytology

Most of our material is bronchial secretion or washings. In the past we have shown that examination of sputum for tumor cells can be most useful and recently have had several positive cases. However, care must be taken in obtaining a satisfactory specimen for submission. Bronchial washings usually yield a higher percent of positives.

In the past year we examined 175 different cases with 226 separate examinations. Of these 18 or 8% were positive and 90% of these constituted the initial means of diagnosis. Naturally most of the cases in which cytology was done were subsequently shown not to be carcinoma. In any series of bronchogenic carcinoma bronchial washings will yield up to 90% positives. This procedure is most useful in inaccessible endobronchial lesions which cannot be biopsied. There are three chief sources of false positive diagnosis. Previous bronchoscopy, radiation to the chest, and squamous metaplasia usually associated with bronchiectasis, can on occasion produce cells which simulate carcinoma.

Effusions

The two commonest sources are pleural and ascitic fluid.

Last year there were 137 different cases with 174 examinations of which 44 (25%) were positive for carcinoma. Over 80% of these constituted the initial and only means of diagnosis (excluding autopsy confirmation of diagnosis). Examination of an effusion for tumor cells is most valuable, but it also is the most difficult field in interpretive cytology. Because of this false positives and false negatives tend to occur. In benign effusion there is a proliferation of the mesothelial lining cells and these can and do reduplicate all the features of carcinoma cells. For this reason it is important to have good clinical data when attempting to interpret the material. A few years ago we saw the case of a 14 year old girl who, on the basis of cells found in ascitic fluid, was diagnosed as having carcinoma of the ovaries. This patient was referred to the Winnipeg General Hospital from another province for x-ray treatment. The radiotherapist was skeptical of the diagnosis and the slides were requested. I considered the cells present not to be carcinoma. Laparotomy subsequently showed the effusion to be due to cirrhosis of the liver.

Since cells undergo autolysis rather rapidly, all effusions removed must be fixed as soon as possible. An anti-coagulant should always be used to prevent clotting and if the entire specimen is to be submitted a preservative such as formalin should be added. If laboratory facilities are available the fluid can be centrifuged and the sediment smeared on glass slides and the remaining "clot" fixed in the centrifuge tube.

Miscellaneous Sites

Gastric washings, oesophageal washings, urines, etc. in selected cases are all worthwhile. The value of any material depends mainly on the care taken in the collection and submission since most of these sites require special care in collection.

Worthy of special mention is the examination of secretion from a discharge from breast nipple.

This can be most useful and should be used more. A few drops of secretion are placed on a glass slide, smeared and air dried.

Aspiration Biopsy

This procedure in my opinion should be much more widely used. It is a relatively simple procedure which generally speaking can readily be done in an office. Last year we did only 41 aspiration biopsies, and in those subsequently proven to be malignant the aspiration biopsy was positive in well over 90% of the cases.

It has often been stated that aspiration of a tumor is dangerous because it will spread the tumor. Memorial Hospital in New York now has twenty years experience with this procedure and they conclude that there is no evidence that this happens. This procedure is especially valuable in breast lesions and in suspected metastatic carcinoma in lymph nodes. Aspiration of a tumor usually provides sufficient material for both a smear and a "clot" — which can be fixed and sectioned like a small biopsy. Classification and grading of tumors is often possible.

Examples of Cases Showing Problems

A review of the cases seen last year points up certain advantages and problems of this technique:

Case No. 11026 — consulted a doctor because of vaginal spotting. The patient was under 30. Visual and manual examinations of the cervix were negative. Biopsy and smears were taken. The smears were positive, the biopsy negative. Because of the necrosis in the smear it was suggested that an endocervical biopsy be taken. This showed invasive carcinoma.

2736/55— This patient had a smear reported as positive in Chicago. A single small routine biopsy showed a small focus of in situ carcinoma. The subsequent cone biopsy of the cervix was negative and follow-up smears have remained negative. This case is the third I have seen where the biopsy cured the patient, i.e., the lesion was so small that biopsy entirely encompassed it.

1247/57— Routine cervical smears were reported as positive. Patient was then admitted for a dilatation and curettage. Following the scraping of the endometrial cavity and the endocervical canal a cone biopsy was done. Sectioning of this material showed an absence of surface epithelium which had been denuded by the previous procedure. No diagnosis was then possible. Several months later the patient again showed atypical cells. In taking biopsies care must be taken not to scrape off the surface epithelium. A dull biopsy forcep slipping over the surface of the cervix can ruin the specimen for interpretation.

Post Irradiation in Carcinoma of the Cervix

We have now seen a number of cases where a routine post radiation smear was positive and biopsy negative. In the presence of slough and necrosis biopsies are often negative, and because of stenosis and contraction of the vault obtaining a biopsy may be difficult.

Smears are an excellent means of following post-irradiation cases.

375/57— smears showed numerous abnormal cells. An infected endocervical polyp was found and removed. Subsequent smears were normal.

10925/56 — plural effusion — two aspirations were negative for tumor. Thoracotomy showed a large sarcoma. Sarcomas rarely shed tumor cells into an effusion whereas carcinomas always do.

6269/56— a 24 year old girl with a unilateral pleural effusion. Six aspirations of this fluid were examined and no diagnosis was possible on the basis of cytology. Two years later this patient had a diagnosis of Hodgkins disease proven by biopsy.

Effusion due to Hodgkins and lymphosarcoma can only occasionally be diagnosed.

Summary

The use of cytology, especially for the diagnosis of carcinoma is now a well accepted procedure. Adequate facilities for interpretation are available and I believe that this procedure could be more used with advantage to patients and physicians. This procedure is particularly valuable in the diagnosis of early non-invasive cervical carcinoma, in detecting endobronchial tumors, and in examining effusions for carcinoma. Further use should be made of aspiration biopsy techniques.

Surgery

Fundamental Factors in Biliary Tract Disease

Based on a Review of 487 Consecutive Cases

A. C. Abbott, F.R.C.S. (Ed & C.) F.A.C.S.
J. T. MacDougall, F.R.C.S. (Ed. & C.)
T. K. Goodhand, M.D.
K. D. McKenzie, M.D.

Biliary tract disease is a very common ailment. It is frequently associated with lesions in neighbouring organs, particularly the pancreas, duodenum and stomach. One would be very remiss, however, to omit coronary artery disease. The symptoms in many patients are so classical that the diagnosis is almost self-evident. These require a minimum of investigation to make the diagnosis. In other cases, the symptoms are vague and bizarre, and require painstaking investigation before other associated pathology is recognized, and a complete diagnosis is established. An easy and quick diagnosis of gall stones is no excuse for omitting a complete examination. It is extremely embarrassing to open the abdomen of a patient and find a secondary carcinoma of the liver from an unsuspected lesion of the colon, or a posterior perforating duodenal ulcer.

Cholecystectomy in recent years, has become a very common surgical procedure. In some cases it is a very easy operation. In a fair proportion of cases, however, it is associated with considerable hazard, especially in those of advanced years. As a result, we feel that the story of our experience during the past ten years may be of some value.

In the past decade, we have operated on 487 cases of biliary tract disease. Table (I). It will be noted that the ratio of females to males is about 4 to 1 in the total series. As age progresses, however, the incidence in the male increases and reaches parity in the older age group.

TABLE I

Biliary Tract Surgery
A Review of 487 Consecutive Cases

				%
Patients			487	
Males			106	21.8
Females			381	78.2
	Age Distribution			
	Male	Female	Total	
20 - 29	1	39	40	8.2
30 - 39	8	65	73	15.0
40 - 49	22	103	125	25.6
50 - 59	35	103	138	28.4
60 - 69	21	59	80	16.4
70 - 79	12	16	28	5.8
80 - 89	2	1	3	0.6

From:
1. The Department of Surgery, University of Manitoba Medical School.
2. The Abbott Clinic, Winnipeg.
3. Read at The Manitoba Medical Association Meeting, October 16, 1957.

The signs and symptoms are illustrated in Table (II). These figures are almost an exact replica of those published by the Lahey Clinic[1], Table (III), in a much larger series of cases.

TABLE II

Signs and Symptoms in 487 Cases

	Number	%
Upper abdominal pain and colic	421	87.5
Previous attacks	385	79.0
Associated nausea and vomiting	246	48.7
Jaundice or history of jaundice	87	18.8
Dyspepsia	248	63.5

TABLE III

Signs and Symptoms of Cholecystitis and Cholelithiasis

	Cases Number	%
Pain	1,187	87.5
Belching and Flatulence	861	63.5
Nausea and Vomiting	661	48.7
Jaundice	255	18.8
Fever	165	12.1
No Symptoms	29	2.1

Table (IV) tells the story of the surgical findings in 87 non-visualizing gall bladders. You will note that 3.4% were normal.

TABLE IV

Pathological Findings in 87 Non-Visualizing Gall Bladders

	Number	%
Chronic Cholecystitis With Stone	59	67.9
Chronic Cholecystitis (No Stone)	6	6.9
Cholesterolosis	2	2.3
Acute Gall Bladder With Stone	9	10.4
Acute Gall Bladder (No Stone)	3	3.4
Hydrops With Stone	2	2.3
Hydrops (No Stone)	1	1.1
Carcinoma — No Stone	2	2.3
No Pathology	3	3.4
	87	100.0

TABLE V

Type of Incision in 487 Consecutive Cases

	Number	%	Last 218 Cases
Paramedian	161	33.1	21
Trans. muscle split	195	40.0	157
Trans. muscle split extended	28	5.7	25
Trans. incision	81	16.6	14
Kocher incision	21	4.3	1
Kidney incision	1	0.4	0

In investigating a patient for gall bladder disease, great reliance is placed on gall bladder visualization. We have covered this subject quite thoroughly in a previous paper[2], but the subject of the non-visualizing gall bladder is worthy of mention. A second visualization should always be done, if no visualization is obtained at the first trial, in many cases using more dye. This second investigation will prevent some errors.

TABLE VI

The Mortality in Acute Biliary Tract Surgery in Various Age Groups in 49 Cases

Acute Cholecystitis:	Up to 49 years No.	Deaths	%	50 - 64 years No.	Deaths	%	65 years and over No.	Deaths	%
Cholecystectomy	16	0	0	13	0	0	4	1 (69 years)	25
Cholecystostomy	1	0	0	1	0	0	4	1 (74 years)	25
Cholecystectomy and Choledochotomy	1	0	0	6	0	0	3	0	0
Total	18	0	0	20	0	0	11	2	18.2
Final Total	49 Cases — 2 Deaths — 4% — Ages 69 - 74								

	%
38 Cases between 20 - 64 — No Deaths	0
11 Cases between 65 - 74 — 2 Deaths	18.2

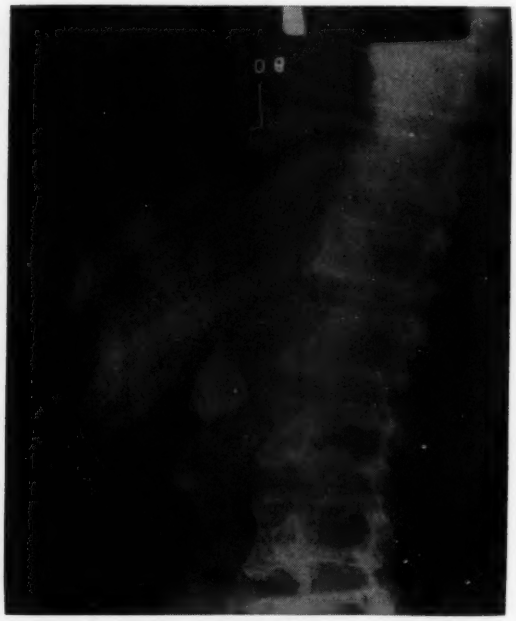


Figure 1

The gall bladder is visualized and the large shadows of stones in the gall bladder are intensified by impregnation of dye. The two small shadows below suggest stones outside of gall bladder or in a diverticulum.

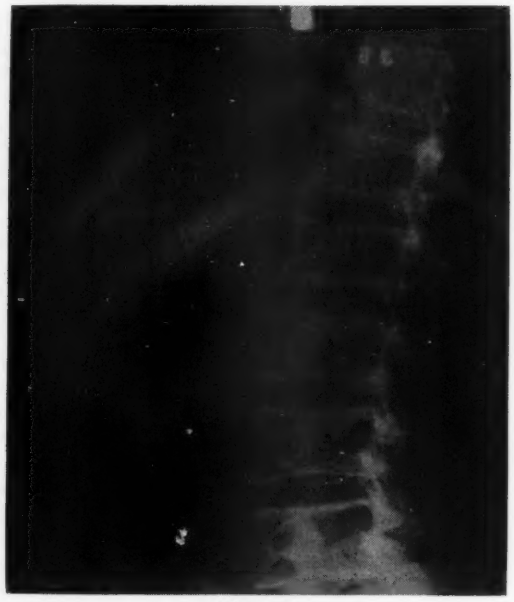


Figure 2

Intravenous cholangiogram showing common duct visualized and somewhat enlarged. The stones in the gall bladder are quite well visualized.

Figure 3

The gall bladder is greatly thickened. One mixed stone and one pigmented stone lies in the gall bladder. Two small mixed stones were present in the pouch at tip of the gall bladder.

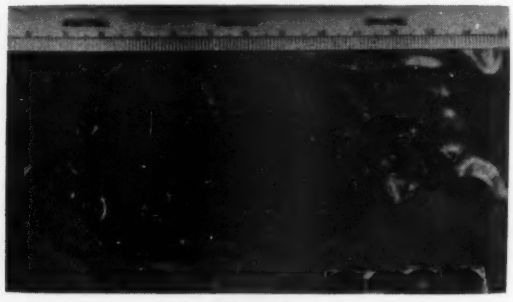


Figure 4

Two stones can be seen lying in the pouch at the tip of the gall bladder. No dye entered the pouch in several visualizations.

In some cases the gall bladder visualization is not conclusive. Figures 1 and 2, a combination of gall bladder visualization and intravenous cholangiography shows stones inside, and two stones outside the gall bladder shadow. A diagnosis of diverticulum of the gall bladder was made. The pathological lesion found in the gall bladder at operation is shown in Figures 3 and 4. Twelve stones were also removed from the common duct. Post operative result was excellent.

The diagnosis has been made, the decision for surgery decided upon, all investigations are completed and the patient is draped upon the operating

table. What incision will you use? Undoubtedly many factors, such as the presence of pathology in some other organ, the stature of the patient, or the laxity or thickness of the abdominal wall, will influence your decision.

During the last ten years, we have used the various incisions shown in Table (V). In a previous ten-year series, the Kocher incision was a favourite. It gives wonderful exposure, but is less anatomical than the paramedian or transverse incision. We gradually adopted the paramedian incision, and from that came to the formal transverse incision. About five years ago, we began using a transverse muscle splitting approach, and now use it in approximately 75% of all our cases. We like this incision. It does, however, require a very definite technique. The incision is made exactly the same as the transverse incision through the skin and subcutaneous fat down to the rectus muscle medially and the external oblique laterally. The outer sheath of the rectus is divided completely to the mid-line. The rectus muscle, instead of being divided, is mobilized and retracted medially. The external oblique, transversus, and internal oblique muscles are split in line with their fibres, and carried well laterally. Any presenting nerves are carefully dissected out and displaced either upwards or downwards. The fascia transversalis and the peritoneum are then divided transversely in line with the incision, and the gall bladder is exposed.

With modern anaesthesia and the use of muscle relaxants, relaxation is quite sufficient for good exploration of the abdominal cavity. The biliary tract is then exposed in a very definite manner. Never less than three packs are used. The first pack walls off the stomach, the second isolates the colon and small bowel, and the third controls the duodenum. Broad Deaver or Kelly retractors are inserted and are rarely shifted during the operation. This gives beautiful exposure of the entire biliary tree. It is essential to have a pack on the duodenum with downward retraction, putting the common duct on the stretch. It makes dissection as easy as you would do it in the dissecting room. Protrusion of bowel into the wound is very unusual, thanks to our very excellent anaesthetic department. The incision is also very flexible as it can be converted, if more room is required, into a formal transverse incision, by simply dividing the rectus muscle. As you will note in Table (V) we have used this simple transverse muscle splitting incision 157 times, in our last 218 operations. We have found it expedient in another 25 cases, to divide the rectus muscle, partially or completely, transforming it into a formal transverse incision. There are three musts in this procedure, good anaesthesia, good assistants who stay put, and a knowledge of where and how to put in your packs. We feel sure it is the easiest on the patient, and the least traumatic. It is also the most anatomical. We would like to emphasize one fact that you must never forget. In many cases, due to traction, the common duct runs transversely across the abdomen. It looks like the cystic duct. Never put a clamp on any structure until you have all the structures visualized. The right hepatic artery frequently presents and resembles the cystic duct. In many cases it lies in the gall bladder bed, and the cystic artery is a very short structure, frequently not more than 1 cm. or less in length. Unless you completely visualize these structures, your troubles will be major ones.

The mortality rate of any operation is always of great importance, not only to the surgeon and the referring physician, but most of all to the patient. On occasion, no matter what the risk may be, it must be faced. Let us consider the mortality rate at various ages in acute and chronic disease of the biliary tract.

Acute Cholecystitis

In the past decade, many surgeons have come to the conclusion that an acute gall bladder is best treated surgically, if seen within reasonable time, and there is no definite contra-indication to surgery. In the great majority of cases, time can be allowed, usually 24 hours, to restore fluid balance, do a thorough examination of the blood chemistry, a chest plate, electrocardiogram, and become thoroughly conversant with the general condition of the patient. In this waiting period, it is not uncommon to decide against emergency surgery because of marked improvement in the patient's condition. We have had a more or less flexible rule of operating upon patients in the first 72 hours if there are no contra-indications. Table (VI) illustrates our results. It will be noted that in our small series of cases, little trouble has been experienced up to the age of 65, no deaths having occurred in this series. In the older group, however, the mortality is high, and in our very small series of cases was 18.2%. The number is too small to be of any statistical value.

As a result of this survey, it might be advantageous to reconsider our position on this procedure. Twenty years ago an acute cholecystitis was a medical problem. Occasionally a cholecystostomy was done under local anaesthesia. Then an interlude occurred in which many surgeons felt that an acute gall bladder should be treated the same as an acute appendix. The pendulum is now undoubtedly swinging back to more conservative treatment. In 1954 Doubilet, Reed and Mulholland[3] published an excellent report on The Medical Treatment of 116 Cases. Acute inflammation, a tender mass in the right upper quadrant, peritoneal irritation, fever and leucocytosis, were present in all. Nineteen had an associated pancreatitis. One died with chest complications. One had a subphrenic abscess but recovered.

A very good working rule is to insist that all patients with acute gall bladders be hospitalized. Their subsequent course is unpredictable. Energetic medical care should be instituted including correction of dehydration, sedation, nasal suction, antibiotics, antispasmodics, and a rapid investigation. The great majority will subside. In a lesser number, all symptoms at first regress and then reappear. In these cases, surgery is probably indicated. In about 20% symptoms are progressive and in these cases we advise surgery.

We prefer to send a patient home, who has recovered from an attack, for a period of 6 to 8 weeks, if they are in a position to return quickly if they have a recurrence. Such a recurrence is not unusual. Many surgeons with wide experience disagree with this latter program and operate before sending them home. They feel recurrence of symptoms is too common.

When we turn to the surgical treatment of chronic disease, the picture is much different. In these cases you have ample time to carefully assess your patient, treat any defects in their circulatory or respiratory systems, and in general have them in good condition before admitting them to hospital. We have an absolute rule of never admitting a patient for any major procedure one day and operating the following day. This is particularly true of married women, who always enter hospital tired. Two or three days rest before operation is the best way of avoiding mortality and morbidity that we know of. Any departure from this procedure is, in our opinion, akin to malpractice. In our series of 428 cases, Table (VII), operated upon for chronic lesions of the biliary tract, we had 4 deaths, a percentage of .94. You will note we had no fatalities under age 50 in 216 consecutive cases. This included 60 cases in which concomitant surgery was carried out on the common duct. In 153 cases between the ages of 50-64, our mortality was 1.3%. One death occurred in a woman age 63, following a cholecystostomy. The other death was in a woman age 62, with severe asthma, following removal of a stone from the remnant of a cystic duct.

TABLE VII

Mortality Rate in Chronic Biliary Tract Surgery in Various Age Groups — 428 Cases

Chronic Cholecystitis:	Up to 49 years No.	Deaths	%	50 - 64 years No.	Deaths	%	65 years and over No.	Deaths	%
Cholecystectomy	156	0	0	104	0	0	38	2 (69/79 yr.)	5.3
Cholecystostomy	0	0	0	1	1 (63 yr.)	100	2	0	0
Cholecystectomy and Common Duct	54	0	0	41	0	0	17	0	0
Common Duct only	4	0	0	3	0	0	1	0	0
Choledocho-duodenostomy Stricture	0	0	0	1	0	0	1	0	0
Cystic Duct Stone	0	0	0	1	1	100	0	0	0
Sphincterotomy	2	0	0	2	0	0	0	0	0
Totals	216	0	0	153	2	1.3	59	2	3.4
Final Totals	428 Cases — 4 Deaths — .94%								
Associated Common Duct Surgery	20 - 49 years — 27.7%								
	50 - 64 years — 30.6%								
	65 - 79 years — 32.2%								

Common Duct Stones

Autopsy and clinical reports in recent years have suggested that common duct stones are present in 20% of all patients with cholelithiasis. The incidence increases with age. It is suggested that in 100 cases in which stones are recovered, 40% will have jaundice at the time of surgery, 20% a history of preceding jaundice and 40% will have never been jaundiced.

In a paper published 2 years ago,[2] we reported exploring the common duct 96 times in a series of 360 cases or 26%. Stones were recovered in 35 cases, or 9.7%. This compares favorably with a series reported by the Lahey Clinic,[1] with a recovery rate of 10.1%.

In 1954, Hallenbeck, Walters, Gray, Priestly and Waugh[5] operated on 1036 gall bladders; 93% contained stones. Cholecystectomy was done in 769, and cholecystectomy and choledochotomy in 246. In this series of 246 cases of 23.7% of the total number, stones were recovered in 95 or 38%, and 8.2% recovery rate for their total series. Between 8 and 10% recovery rate is the average rate reported in the literature. Some, however, are reporting as high as 18% by using routine cholangiograms on the table.

This brings up the question of operative cholangiograms. We have not been carrying out this procedure routinely. It adds at least ½ hour to the time of the operative procedure, possibly adds to the morbidity, increases the cost to some one in the operating room charges, and is in its present stage not 100% accurate. We are not unfriendly, however, to this procedure, and feel it is a welcome and a valuable aid.

In our last 118 cases, of biliary tract surgery, we explored the common duct 34 times or 28.1% and recovered stones in 11.1%. In our complete 10-year survey of 487 cases, we know or suspect missed stones in 7. In 2 we have cholangiograms showing residual stones in the hepatic ducts within the liver. Missed stones must be considered as morbidity.

Is mortality increased by exploring the common duct?

It has been generally conceded that cholecystectomy, combined with any operative procedure on the common duct, carries with it an increase in mortality. We have consistently found otherwise. You will note in Table (VIII) that we have carried out operative procedures on the common duct 136

TABLE VIII

A Comparison of Mortality in Simple Cholecystectomy — Cholecystectomy and Common Duct or Common Duct Only

	Up to 49 years				50 - 64 years				65 and over			
	Cholecystectomy		Cholecystectomy and Common Duct or Com. Duct only		Cholecystectomy		Cholecystectomy and Common Duct or Com. Duct only		Cholecystectomy		Cholecystectomy and Common Duct or Com. Duct only	
	No.	Deaths	No.	Deaths	No.	Deaths	No.	Deaths	No.	Deaths	No.	Deaths
Acute	16	0	1	0	13	0	6	0	4	1	3	0
Chronic	156	0	60	0	104	0	47	0	38	2	19	0
Totals	172	0	61	0	117	0	53	0	42	3	22	0

times, without any mortality. These procedures include exploration and removal of stones, resection of strictures and sphincterotomy.

In 329 simple cholecystectomies, our mortality was 3 or .91%.

In all surgical procedures, we must consider not only mortality, but morbidity. In going over our records, results which are shown in Table (IX)

TABLE IX

Comparison of Morbidity at Various Ages Measured by Days in Hospital

Operation:	Up to 49 yrs.	50-64 yrs.	65 and over
Cholecystectomy	14.4	15.2	14.1
Cholecystectomy and C.D.	22.3	23.0	21.3
Average	16.5	17.2	16.4

Total Average — In Days

Cholecystectomy	14.4
Cholecystectomy and C.D.	22.3
Average	16.5

we find that the average time in hospital for simple cholecystectomy was 14.1 days. This includes the time that the patient was in hospital, from the day of admission to the day of discharge. You will also note that the average time in hospital for common duct surgery was 21.3 days. There is little difference in days spent in hospital in the three age groups.

Patients admitted for cholecystectomy and common duct exploration frequently require many more days pre-operative care than those admitted for simple cholecystectomy. We do routine post-operative cholangiograms before we discharge the patient. This lengthens the time of hospitalization.

Complications:

Table (X) illustrates the complications we encountered in our series of cases. In a previous publication[2] we listed all cases in which we encountered prolonged drainage post-operatively as subphrenic or subhepatic abscesses. We have revised this classification, allocating only those cases in which we had to resort to further surgery as subphrenic abscess. We encountered 2 cases in which we had drainage of duodenal content. Both were associated with the use of long armed T-tubes. We are not entirely satisfied with them.

In three cases following sphincterotomy we finally removed, surgically, a plastic indwelling catheter left in the common duct. One was removed after 10 months. All were removed through a small incision in the upper loop of the jejunum from the left side. We have buried tubes in the common duct many times for many years, and have never encountered this trouble with ordinary rubber tubes.

Figure (5) illustrates a large dilated common duct due to a stricture of the sphincter of Oddi, which was treated by sphincterotomy. Figure (6) shows the catheter following removal. It was quite patent.

In one case we injured the common duct. This patient was mildly jaundiced for months before operation. At the time of operation a small common duct was found. It was aspirated and clear bile obtained. No stones were palpable. It was carfeully preserved and not opened. The gall bladder with stones was removed. Following operation she became jaundiced. There was no biliary discharge. She was re-explored and her common duct could not be found. The liver was explored with a needle and a large duct full of bile located. The duodenum was mobilized and sutured over a tube as a mold, to the under surface of the liver. The patient recovered.

Since compiling this report, we have had a similar case. The common duct was widely dissected and not opened. It was carefully covered

TABLE X

Non-Fatal Complications in 487 Cases of Biliary Tract Surgery

	Patients Up to 49 years		Patients 60 - 64 years		Patients 65 and over			
Complication:	G.B.	G.B. & C.D.	G.B.	G.B. & C.D.	G.B.	G.B. & C.D.	No.	%
*Prolonged drainage — 3 subphrenic abscess	2	3	2	3	1	2	13	2.6
Wound infection	2	3	2	3	2	0	12	2.4
Pulmonary complications	3	1	3	2	1	0	10	2.0
†Duodenal fistula	0	1	0	0	1	0	2	
Thrombophlebitis	2	0	0	2	0	2	6	1.2
§Post-op. hernia	1	3	0	0	2	0	6	1.2
Wound disruption	0	1	0	0	0	0	1	
Secondary haemorrhage	0	1	0	0	0	0	1	
Shock — severe	0	0	0	0	0	1	1	
Liver abscess	0	0	0	1	0	0	1	
Cardiac complications	0	0	2	1	0	0	3	0.6
Mental confusions, etc.	0	0	0	1	0	0	1	
Injury to common duct	1	0	0	0	0	0	1	

* 3 required operative drainage.
† associated with use of long arm T-tube.
§ 1 Kocher incision — 3 transverse incisions — 2 transverse muscle split.

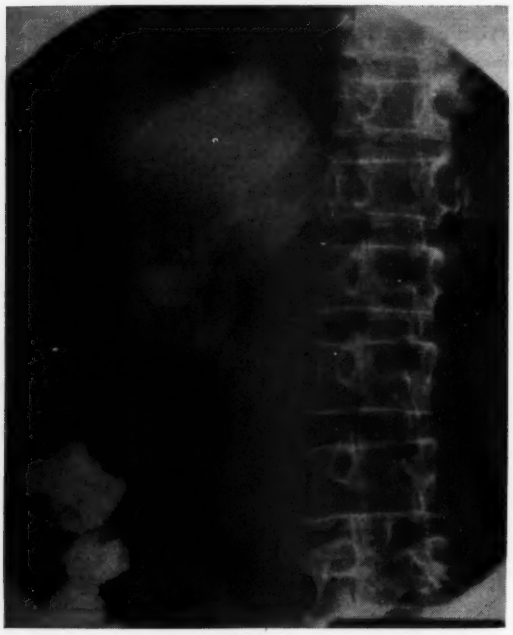


Figure 5
Note great width and lengthening of the common duct, due to narrowing or spasm of sphincter of oddi.

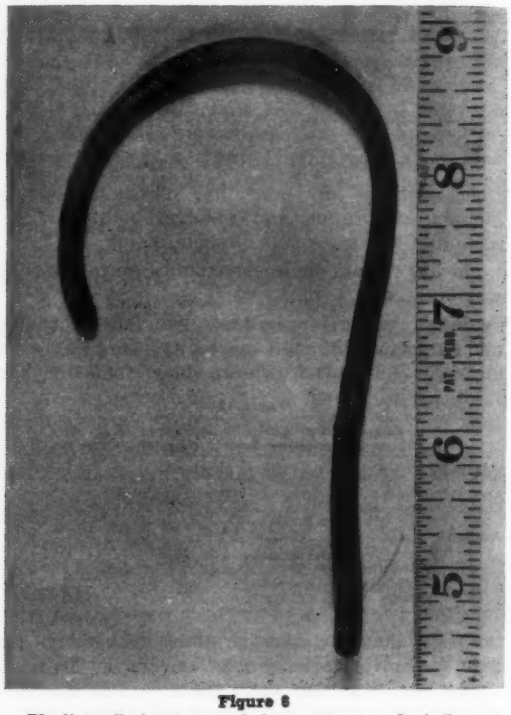


Figure 6
Plastic catheter removed from common duct through upper loop of jejunum.

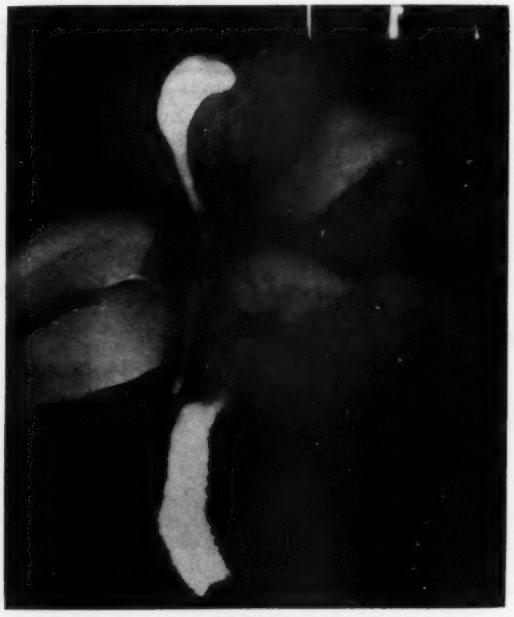


Figure 7
Normal biliary tree of dog visualized post-mortem.

Figure 8
Post-mortem cholangiogram of biliary tract of dog, showing stricture of common hepatic duct just above the cystic duct. Note dilatation of duct above stricture. The mucosa was intact.

over with peritoneum at the termination of the operation. She became jaundiced after the penrose drain was removed and a profuse biliary discharge developed. She was explored a few days later. A stump of common duct ½ inch, was located. Bile was pouring out of the sloughing end of the cystic duct. A choledochoduodenostomy was done. She made an uneventful recovery.

In this case, we are sure we did not crush or clamp the common duct. We believe, however, that we injured its blood supply by wide dissection, and necrosis followed. Appleby[5] has done a great deal of research work on this subject and is firmly convinced that necrosis from interruption of the blood supply is the cause in these unfortunate cases.

We have been investigating this problem ourselves, by stripping the biliary ducts in dogs. We have, in a limited number of cases, been unsuccessful in producing necrosis of the bile duct, but have produced a stricture. Figure (7) is an actual cholangiogram of the biliary system of a dog. You will note how rapidly the dye pased into the duodenum. Figure (8) is a post-mortem cholangiogram from a dog in which the biliary tree had been stripped 3 weeks previously. You will note the definite stricture formed in the common duct just above the cystic duct. The small amount of dye that has passed into the duodenum is also confirmation of stricture. The intra-hepatic ducts are dilated. The mucosa was intact, the stricture being limited to the outer portions of the wall.

Summary:

1. The results in 487 consecutive cases of biliary tract disease are reported.

2. The value of careful, complete investigation before surgery is emphasized.

3. Revisualization of gall bladders not visualizing on the first attempt, is indicated. The findings in 87 such cases are reported.

4. The value and feasibility of a muscle splitting incision is presented. The use of abdominal packs is advised as a method of giving one clear vision throughout the surgical procedure.

5. The treatment of acute and chronic disease of the biliary tract is discussed and the mortality at various ages shown.

6. Exploration of the common duct increases the stay in hospital, but does not increase mortality.

7. The complications in biliary tract surgery are discussed. An explanation of why common ducts are apparently injured is offered. This should be thoroughly investigated. Experimental proof is advanced for the author's ideas.

Bibliography

1. Colcock, Bentley P. and McManus, J. E.: Cholecystectomy for Cholelithiasis — A Review of 1,356 Cases. The Surgical Clinics of North America, 35: 765-771, June, 1955.
2. Abbott, A. Clifford, Goodhand, T. K. and MacDougall, J. T.: Biliary Tract Disease — A Review of 369 Consecutive Cases. Canadian Medical Journal, 25: 395-400, 1956.
3. Doubilet, H., Reed, George and Mulholland, J. H.: Delayed Operative Management of Acute Cholecystitis. J.C.M.A., Vol. 155, 18: 1570-1573, August 28, 1954.
4. Hallenbeck, George A., Walters, Waltman, Gray, Howard K., Priestley, James T. and Waugh, John M.: Report of Surgery of the Biliary Tract and Pancreas for 1954. Proceedings of the Staff Meetings of the Mayo Clinic, 30: 640-646, December 28, 1955.
5. Appleby, Lyon M.: Personal Communication.

Closed Intra-Abdominal Wounds

H. L. McNicol, M.D.
Flin Flon, Manitoba

Serious or even fatal intra-abdominal wounds may occur from what appears to be comparatively mild injuries to the abdominal wall with little or no evidence of external trauma. They can be classified grossly into those which would recover without any treatment, those that would die regardless of treatment and those that would die unless treated properly; also into those in which the chief difficulty is hemorrhage and those in which the main injury is rupture of an intra-abdominal viscus.

I am confining myself entirely to closed abdominal wounds with no other injury.

Early diagnosis and treatment are necessary to reduce morbidity and the loss of life. We, as general practitioners, are frequently the first medical people to see these patients. It is our first duty to save the patient's life and secondly to return the patient to as nearly a normal state as possible.

The patient may be presented with very little apparent evidence of violence to the abdominal wall or what may seem to be only a trivial injury. There may be very little evidence of shock, or on the other hand, he may be in a severe state of shock. Any patient presenting himself to a physician because of discomfort due to a blow to the abdomen deserves very close observation. If perchance, the patient is in a state of shock, this person must be treated for shock, while we are making the diagnosis. There are a number of aids that may be used in making the diagnosis, such as x-ray, hemoglobin and hematocrit estimations, urinalysis, aspiration of the peritoneal cavity, but most important of all aids is close, intelligent observation by the doctor who is in charge of the patient. The first requisite is complete physical examination, looking at the patient as a whole, which includes facial appearance, pulse rate and character, blood pressure, respiratory rate and character. The abdomen must be assessed; going right back to the fundamentals, that is observation as to contour, movements with respiration, area of local tenderness and whether or not there is any local guarding. The stethoscope, which is too frequently forgotten, is used to establish the presence or absence of bowel sounds. At this stage we must examine the hernial orifices and inquire about the possibility of hernia. I have seen a patient's life lost because this was neglected.

Now that we have established a physical base, we can proceed with various tests. The tests of most value include x-ray for the presence of free air, areas of increased density, or displaced stomach, colon, etc., hemoglobin estimation, hematocrit estimation and urinalysis. Aspiration of the abdominal cavity for blood or bowel contents is of value if positive, but in my opinion is a poor method of establishing a diagnosis. Any of these methods are good only if positive.

Now we must watch for developments. It is a fact that with either perforation or hemorrhage, time is a great factor, but intelligent, watchful waiting can be of great value in establishing a diagnosis and deciding the method of treatment.

If the area of local tenderness and muscle resistance is extending, bowel sounds decreasing or absent and possibly increasing rectal tenderness, the likelihood is that there is a perforation of a hollow viscus. If on the other hand there is increasing abdominal distention, with rising, compressable pulse and falling blood pressure and falling hemoglobin, with possibly diaphragmatic irritation as evidenced by shoulder pain, the likelihood is that this is hemorrhage. If either condition is diagnosed or the patient is deteriorating, exploratory laparotomy is a must.

I repeat, the only sure method of assessing the patient is continuous and intelligent observation by ourselves, not by a nurse or a junior intern. Most patients can be safely observed for one to two hours. Preparation of the patient for operation requires intravenous therapy in the form of electrolytes and/or blood. There should be special mention made of one particular intravenous solution, that is dextran. When it first became popular, the indications were that it could be used in great quantities, but recently there have been several articles maintaining that it increases the bleeding tendency if used beyond 1,000 cc's. A good rule is to have a minimum of three pints of blood available.

The patient must be given pre-operative sedative. The patient's general condition must govern the amount. This can be given intravenously, intramuscularly or subcutaneously, depending upon the time element. Many trained anaesthetists prefer the intravenous route. Pre-operatively the stomach should be emptied, by means of a levine tube or if food is known to be present an Ewald tube.

The choice of anaesthetic is one that will have to be decided upon on its merits, with two exceptions, never local and never spinal anaesthesia. The ideal is intratracheal general anaesthesia with the use of some relaxing agent. If you have no trained anaesthetist, I prefer open drop ether as being the safest anaesthetic agent.

The abdominal wall must be thoroughly scrubbed preferably after the patient is asleep, using phisohex or a similar soap and water. This may seem to be a minor contribution, but also may save many sleepless nights, both for the patient and the doctor by preventing the development of infection.

The incision is a somewhat controversial point and has to a lot with individual preference, but if

Given at Second Scientific Convention, College of General Practice of Canada, Winnipeg, April 14, 15, 16, 1958.

the diagnosis is in doubt, my preference is to make a para-umbilical incision on the side that physical examination would indicate the lesion. This incision can be extended up, down or sideways as is warranted by the occasion. One point that is frequently forgotten is that cases with gross intra-abdominal bleeding go into a deeper state of shock when the peritoneal cavity is opened into and until we have controlled the bleeding.

Now once inside the abdominal cavity, if we see that there is no gross or very little bleeding, with a comparatively small amount of soiling, our worries are just beginning, for we must search for what has made us bring the patient to operation. This must be done in an orderly fashion, starting first at the stomach and continuing down the gastro-intestinal tract, until we find the lesion. As both the anterior and posterior wall of the stomach must be looked at we must open into the lesser peritoneal sac. If there is a hole in the stomach, it is simply a matter of closing this, with whatever suture material you prefer to use. We use chromic catgut for the mucosa and silk for the other layers. It should be closed in either two or three layers. While in the lesser sac, the pancreas should be examined thoroughly, because on superficial examination, there may be what just appears to be a hematoma around it, but on closer examination it may be found to be fractured. If this is so, there are two methods available in dealing with the condition. The one is simply to suture the ends together. Some people even try to find the ducts and suture them, but this may be time consuming and not too good in a patient who is already in a critical condition. The second method is to excise the distal end of the pancreas by itself or along with the spleen. Although I have never done this, post-operative results are very good with little or no instance of diabetes. The remaining end of the pancreas is oversewn. This area should always be drained.

The next examination is that of the duodenum. Injuries to the duodenum are notoriously silent and treacherous, both from the standpoint of diagnosis and treatment. There may be very little positive physical findings, except deep tenderness in the epigastrium. The only symptoms the patient may have are some nausea and deep seated epigastric pain. There may be no positive laboratory findings. At operation, there may again be only a visible hematoma and, unless very closely inspected, one may miss a tear and even a complete transection of the duodenum. Here it may be necessary to reflect the peritoneum and examine the posterior surface even cutting through the ligament of Treitz. The treatment is closure of the hole or anastomosis of the ends using only two layers of suture material. An enterostomy may be necessary to overcome possible obstruction.

Now we progress to the small bowel. It seems to be the general impression that there is never a perforation of the small bowel caused by blunt trauma to the abdomen, due to its extreme mobility, but this is a fallacy. The most common part to be involved is the jejunum and upper ileum. However a bursting wound may occur in any part of the small bowel. There may be associated tears or thrombosis in the mesentery. If there is only one tear in the bowel, which is usual, this can be sutured preferably transversely in order not to diminish the size of the lumen. Here we would only use two layers of sutures. If there is more than one tear, or if the mesentery is injured or the blood supply to the bowel is in jeopardy, it may be necessary to do a resection. This may be done by using the open or closed method with end to end, side to end or side to side anastomosis.

The large bowel is dealt with in much the same manner as the small bowel, with the exception that it may be wise to do a proximal colostomy to divert the fecal stream. In dealing with both small and large bowel, it may be life saving to do an exteriorization of the involved area with definitive surgery at a later date. I think it is always wise to drain the peritoneal cavity in cases of perforation of the bowel or injury to the pancreas with the possible exception of a wound only involving the stomach.

If after the incision is made, one sees the peritoneal cavity filled with blood, the most likely organ involved is either the spleen or the liver. Depending upon your preoperative studies the incision will be made on the right or the left side. If one looks at the spleen, and finds even just a subcapsular hematoma, treatment is removal. Here we might digress for a moment to remind ourselves once again of the possibility of delayed bleeding from the spleen and warn anybody who comes in with an injury to the left upper quadrant or the left lower chest and apparently recovers within two or three days, that there is still a possibility of trouble. There is one sign that may be of value in this particular instance and that is supra-clavicular pressure, just lateral to the left sternomastoid insertion. This may produce pain in the left upper quadrant, if there is subcapsular hemorrhage of the spleen. In removal of the spleen it is mobilized, if possible, thus bringing it into full view on the abdominal wall. Then clamp, cut, transfix and doubly ligate the pedicle, not forgetting that there are some moderately good sized vessels at times in the gastrosplenic ligament. If, perchance, there is difficulty in mobilizing the spleen, there is one method that may be of some value, that is to put a rubber covered clamp blindly on the splenic pedicle. This controls the hemorrhage to a certain extent and does not traumatize the pancreas or bowel sufficiently to cause necrosis. Some surgeons approach the spleen by going through the lesser sac and picking up the main vessels along the superior border of the pancreas. While handling the spleen, it must be remembered that it is very friable, and, if any

fragments of it are left behind, they may produce splenic implants which may cause trouble at a later date.

If it is found that the liver is involved, any macerated tissue should be removed. Lacerations of the liver may be dealt with in two ways. One is suturing and the other is packing. As you all know, liver tissue is extremely friable. The two methods of suturing consist of either straight mattress sutures of chromic catgut material, drawing the edges firmly together and avoiding tugging and jerking movements so that the sutures will not cut through. The other method is to run a suture parallel to the wound edges on each side and then place mattress sutures behind these and pull the wound edges together, again remembering to tie the sutures snugly but not tightly.

If one decides to pack, ordinary gauze may be used or preferably gel-foam or one of the similar substances to be left in place. Gauze of course must be removed in forty-eight or seventy-two hours.

While dealing with the liver, it is extremely important to examine the gall bladder and bile ducts. If the gall bladder is torn, the treatment is removal. If the ducts are torn, it will be necessary to repair them and always leave a "T" tube in the common duct. These ducts should be sutured in one layer, using very fine catgut or silk.

Should the spleen and liver be intact, one must look elsewhere for the bleeding. The most likely spot is the mesentery. Any bleeding vessels must be ligated and if it seems necessary, the involved part of the bowel resected. If we still have not found the bleeding point, we must look further. Frequently we hear about contre courte injuries to the brain, but not too much about this state in the abdominal cavity. However, it does occur and I will cite a rather interesting case history as an illustration. It concerns a rather short, fat, middle aged woman who slipped on the kitchen floor and fell onto a footstool, injuring the right upper quadrant of her abdomen. She complained of pain in her lower chest and right upper quadrant and on first examination, it was thought that she had broken some ribs. However, on closer observation, she obviously had some internal hemorrhage, which was thought, in all probability, to be from the liver. After the necessary preparation, she was explored and it was found that the abdominal cavity was full of blood. The liver and spleen were intact. There was no bleeding from the mesentery. The incision was then extended to bring into view the pelvic organs. Examination here revealed a ruptured ovarian cyst. This was bleeding very actively. This emphasizes two points. Don't forget to look for pathological conditions, giving rise to hemorrhage, and secondly, that the hemorrhage may be at a point distant from the actual site of trauma.

Now what to do about excess blood in the peritoneal cavity? Should it be removed or left? My opinion is, that unless there is gross contamination, as would occur with a perforation, the blood should be left. This has been pretty well proven by recent experiments which show that up to 65 per cent of the fluid blood in the peritoneal cavity is returned into the general circulation and that up to 33 per cent of clotted blood is returned into the general circulation. My impression is that blood itself does not account for intra-abdominal adhesions to any extent. I do not think there should be a drain left in the abdominal cavity for hemorrhage only.

Should we use antibiotics in the peritoneal cavity? This is a very controversial point. But I think, that where there is a localized perforation, it is well to use a combination of penicillin and streptomycin at the site.

Postoperative treatment. These patients should almost all have continuous gastric suction for forty-eight hours or longer if necessary. They should be given intravenous fluids, both electrolytes and proteins of at least 3,000 cc's in twenty-four hours as long as the gastric suction is in place. They should be given antibiotics as indicated. It is always necessary to inquire as to the possible sensitivity of the patient to the antibiotics.

Many of these people will become quite distended. I think the old-fashioned treatment of using heat, either in the form of large foments, infra-red or any other kind of heat to the abdominal wall is of value. It is unwise to use enemas too early, that is within seventy-two hours. In my experience they may relieve temporarily, but many times it seems to increase and prolong the distention. These people not infrequently have a bout of diarrhoea after seventy-two hours. Generally speaking, I welcome this because it suggests peristalsis is again active.

If after the the patient goes along fairly well, for three or four days, and then starts to complain of feeling generally miserable and bothered with cramps, one must be on the lookout for an obstruction due to early adhesions and/or infection. These must be treated as indicated by the patient's condition.

To sum up, I would say treat all bumps on the abdomen with respect. Watch them closely and intelligently and do not hesitate to have a look inside the abdominal cavity, both for diagnosis and treatment, and be prepared to deal with any abdominal lesion.

Psychiatry

Psychological Reactions to Surgery

Presented at the Clinical Luncheon St. Boniface Hospital, May 8th, 1958

J. Matas, M.D.

Introduction

There are many random observations on this topic scattered through the literature, but recently more detailed systematic studies have appeared. They have appeared mainly in the Psychiatric literature and I thought it worth while bringing to your attention what is being done. The most detailed study is one originating in Cincinnati done by a team consisting of psychiatrists, social service workers, psychologists and even a philosopher.

An excellent article by Elman, a surgeon, appeared in 1950. In it he makes an appeal to the surgeon to get to know his patient better and to use the rapport established to help the patient beyond the bare requirements of surgery. He starts off by: "Psychogenic factors play an important, often a decisive part in the convalescence of surgical patients, just as they do in all patients." Or as Flanders Dunbar has said: "patients do better when treated as people rather than bodies." These however, are impressions and some of the work I will describe later, sets out to prove such notions.

Elman takes exception to a suggestion by Menninger: "that surgery is a very immediate sublimation of sadistic impulses." He also objects to Helen Deutsch, when she says of a group of medical students on a party. "One young man had the highly humorous inspiration to cut off the tails of all the dogs in the town, later he became one of the most famous surgeons of the world."

It is generally accepted that the patient in his attitude towards an operation fears two things: 1) injury which may include loss of part of his body, 2) loss of life. However, the anxiety is often not proportionate to the actual danger, and this is another factor I will talk about more, later on. It is apparent however, that what an operation means to the individual patient is dependent on many factors, such as: his age, education, situation, his conflicts, his attitudes twoards doctors, hospitals and many other such sociological and personal factors. The psychoanalysts of course, cannot miss indicating that the neurotic patient who demands an operation does so as a means of atonement for deep seated guilt. Nor could Helen Deutsch, an analyst, avoid concluding from what she had learned from patients who had had operations, that in men the operation is a symbolic castration, and in women, a symbolic delivery. Whether these ideas are correct or not, it is a common observation that surgery ranks close to having a baby as a precipitating event for a psychiatric illness. I refer not only to mild immediate depressive reactions, about which little is written, but surgery is also often blamed, by the patient, for beginning a long standing hypochondriacal reaction.

The Surgical Population—Who are they?

Karl Menninger's article "Poly Surgery and Poly Surgical Addiction," gained wide circulation. He emphasized the fact that many types of psychiatric patients demand surgical operation. Helen Deutsch in the article I mentioned above, points out that many of the patients who subsequently undergo analysis, had had many operations before treatment. By contrast, while in analysis, operations were very rare.

Bennett, A. E. and Engel found that 121 female patients culled from a group of 500 psychiatric records had undergone 205 unnecessary operations. Among 29 men, 39 needless operations had been performed.

Zwerling et al of the Cincinnati group, in the study of their 200 patients, came up with some rather startling figures. Only about one in ten were considered normal psychiatrically. More than half of their population were suffering from what is now called character or personality disorders, including alcoholism. Again, rather surprisingly, one in five of their patients was psychotic. The authors tried to explain the high incidence of psychiatric disorder by pointing out that their population is drawn from the lowest socio-economic groups. It is now becoming apparent that the incidence of mental disorder is higher in members of such a group than in the remainder of the population. In addition they point out that admission to hospital and having a surgical illness constitutes severe stress and acts as a precipitating event for a mental disorder.

Another suggestion is that surgical illness may be an outgrowth of mental disorder. They give the example of a patient with a severe passive aggressive character disorder, with strong needs to drive at break-neck speeds and drink immoderately, being admitted with a fractured skull, which occured when he smashed up his car. The psychological condition here led to the necessity of surgical treatment. Another obvious example is a person who under environmental pressure perforates an ulcer. Less clear cut is the person with an anxiety state and somatic symptoms, which are of such a nature that the patient is sent to a surgical ward.

A type of patient whom I see frequently is the woman who is dissatisfied with her role as one. Such a maladjustment shows up in various ways, one of which is pelvic discomfort, and demands for repeated operations.

For a time, in Winnipeg, backache led many people to the surgical wards. I don't know if the

same thing is happening now, as I'm not associated as closely with this type of work, but at one time there was a misguided notion going around that every backache was due to a displaced intervertebral disc, unless otherwise proven. The result was that many people with psychogenic backaches became surgical patients.

Cosmetic surgery is a problem in itself. It has been generally recognized for some time that the patient who applies for cosmetic surgery may be neurotically motivated. This applies of course not to the person who seeks surgical help for a serious, particularly a traumatic deformity, but for the patient who comes on account of some deviation from the normal. However, some of the authors on this subject, advocate rhinoplasty as an adjunct to psychotherapy. I have recently seen two applicants for cosmetic surgery, but these were obviously extreme cases. One was a boy of 19 who had not looked at his face for four years because he thought his forehead was not the shape it should be. He also thought his shadow was deformed. This was probably a schizophrenic patient, as was another young man, who wanted cosmetic surgery to prevent his face from becoming more and more like that of a monkey, which he thought it was doing.

The Immediate Post Operative Psychiatric Reactions

I. Acute Brain Syndrome

As in the post-partum reactions one has to differentiate from those mental reactions which come about as a result of trauma, infection and the anesthetic, from functional reactions. The acute brain syndromes of course are mainly related to physiological factors. It is usual to attribute the delirium immediately following surgery to the anesthetic. Other possibilities are: sedation, hypoxia, fever, toxins and circulatory impairment. Another factor to which attention is being paid now, is electrolyte disturbance, particularly sodium depletion. This reaction as one might expect, is seen more particularly in patients undergoing heart surgery because of their low salt diet.

Titchener et al of the Cincinnati study, which I mentioned, 18 developed an acute brain syndrome. The authors considered signs of psychological disintegration a sensitive indicator of something going wrong systemically. On occasion when the temperature chart, pulse and so on seemed to indicate good progress, the psychological changes would be the earliest ones to augur a turn for the worse. Early signs of delirium they described are: bizarre choice of words, thickening of speech, mild confusion, increased lethargy and slight suspiciousness. For confirmation they use two diagnostic techniques. One of them is holding a blank white sheet of paper before the eyes of the patient and he is asked what he sees. He often describes transient hallucinatory experiences. Another method they close the eyes of such a patient and massage gently. In a positive response the patient commonly sees flashes of light which are interpreted in an hallucinatory or illusory fashion.

They found four groups of patients most likely to develop delirious reactions. The alcoholic person stands a good chance of doing so in response to the anesthetic or because of withdrawal of alcohol. Such a situation is commonly seen here. A person has been drinking regularly is admitted for a minor surgical procedure and within a few days develops a confusional psychosis as a result of alcohol withdrawal. The traumatic patient, because he enters the hospital under delirium or wakens from a coma in one. A third group are the people who are aged and lonely. There seemed to be some relationship between paucity of visitors, and people interested in elderly patients, and incidence of delirium. The fourth group is the elderly patient, who for some reason has been immobilized for a length of time, or who has lost an arm or leg. With early ambulation there is less likelihood of a psychosis occurring than if the patient is pinned down by traction apparatus.

II. Non Toxic Reactions

The frequently mentioned observation that a depression is a common finding after an operation is confirmed by the Cincinnati group. They saw nine psychotic depressions in their 200 patients. Five of these occurred in patients having various forms of cancer. Here again, the factor of lack of closeness with other people seemed to have a bearing on precipitating this illness. In contrast the cancer patients who did not develop a psychosis were found to have retained contact with supportive relatives, friends and hospital visitors. Sutherland and Ohrbach found that the cancer patient very frequently experiences a sense of isolation, guilt and abandonment. The difficulty arises where the abandonment in fantasy is confirmed in reality.

Another group which showed depressions frequently, as reported from Cincinnati is the aged one. Twenty-two, of their forty-five elderly patients developed a depression. Some of these on the usual dynamics of hostility turned inwards, but partly as a result of inability to stand every day stress, perception of defective body image, loss of self-esteem, and impairment of ability to remain active, seen as a threat to self-preservation.

Helen Deutsch's study of her patients in analysis who had had operations, indicates that often an operation acts in the same way as any sudden severe traumatic event. She points out one interesting difference between this neurosis and a similar one precipitated for example by a car accident. One of the characteristics of a traumatic neurosis is repetitious dreams. Following surgery, the dreamer dreams of himself taking charge of doing the surgery rather than a usual re-living the event approximately as it occurred.

The results of surgery of course are not always bad psychologically. Menninger has called attention to the fact that psychoneurotic patients and depressive patients are sometimes promptly cured by an operation whether necessary or not.

Hebb, who is professor of psychology at McGill, has done pioneering work on the effects of interfering with perceptors of environmental stimuli. He has demonstrated that in simultaneously blocking sight, hearing and touch, psychotic symptoms such as hallucinations are commonly produced in normal subjects.

When the visual means of contact with the environment is shut off as a result of an operation, psychosis is often precipitated. This is particularly so when other means of communication are interfered with. For example, we recently had a patient in hospital who became psychotic after an unsuccessful bilateral eye operation. She was a Ukrainian woman who spoke little English. Later, when another operation was found necessary, we arranged for a Ukrainian speaking nurse to be with her all the time and the whole procedure went quite smoothly.

The Late Consequences of Surgical Illness

One of the classic articles in this field was published by Lindman in 1941. He set out to study the psychiatric behaviour of patients post surgery. He deliberately chose a homogeneous group of 40 women not younger than 20 and not older than 55, who were to have abdominal operations. He left out any patient who showed psychiatric disease at the time of the pre-operative interview. The patients were again interviewed from 10 to 18 months after the operation. He found 25 had no new psychiatric complaints.

The 15 patients with complaints had had a free interval of at least three weeks, and then developed a state characterized by sleeplessness, restlessness, agitation, loss of appetite, restriction of activity and irritability. Ten members of this group were distinctly depressed and had thoughts of violence intruding into their thinking. Duration of the condition varied. In two instances the condition was still present 16 to 18 months after the operation. The others subsided within 6 to 8 months.

Tichener et al. of the Cincinnati group also followed their cases 3-6 months after their surgical treatment. The patient, in whom the surgical status had not changed or was worse, was also worse psychiatrically. In the patients who had had a successful surgical result, about one third were improved psychiatrically, one third were about the same and another third worse.

They postulate that psychological flexibility or basically an ability to adjust psychologically is somehow associated with physiological resilience that permits quick recovery from surgical illness. They hold that the opposite is also true, that psychological rigidity is associated with stationary unrecovered surgical conditions.

They found that the surgical experience not only had the power to make the personality difficulties greater or less, but also sometimes changed the type of symptom. For example, neurotic symptoms might replace somatic symptoms. A woman who had complained of dysmenorrhea after a fibroid uterus was removed, began to show anxiety, depression and delusions of persecution. The opposite also occurred, that is, somatic symptoms replacing neurotic symptoms. The shift occurred in another way, that is, neurotic symptoms replacing behaviour disorders. They described a man who gave vent to his rebelliousness by weekend drinking and fighting. He fell from the end of a scaffold and was admitted for a possible fracture of the cervical vertebrae. Following his discharge, he began having attacks of trembling, prominent perspiration and palpitations. He had severe headaches, a startle reaction and frequent nightmares. Behaviour disorders were also seen replacing neurotic symptoms.

The long term consequences of gastrectomy were reported by Browning and Houseworth. They studied 30 patients who had had a gastrectomy using 30 patients who were being medically treated as controls. They set out to test the hypothesis that in these psychosomatic illnessess, if one avenue of expression, so to speak, is removed, another avenue of expression will be found. The gastrectomy group revealed a significant decrease in ulcer symptoms, but this was compensated for by a significant increase in other psychosomatic and psychoneurotic symptoms.

The analysts keep emphasizing the late traumatic effects of operations performed on children. Menninger refers to a case of a circumcision of a three year old boy who suffered a powerful reactivation of the horror and fright connected with this operation 25 years later in attacks of depression.

Flanders Dunbar says that fears aroused by a tonsillectomy for which adequate preparation had not been carried out may have a lifelong hangover in what she calls castration anxiety. By this, she means the fear of punishment which a child has because of sexual impulses towards his mother. She says that this can shift sexual balance in the direction of homosexuality.

Predicting the Course During and Following Operation

I pointed out in the opening paragraph that many feel that there is some relationship between pre-operative anxiety and response during surgery. Herring set out to test this hypothesis. He used a large number of psychological tests pre-operatively and found there was some relationship between some of the tests and response to surgery. His

work was not conclusive, but tests which consistently indicated a great deal of anxiety or fear, and maladjustment for various reasons would corelate with instability during actual operation, as indicated by blood pressure responses. Anxiety, of course is not a fixed quantity of personality and is influenced by the management of the patient in hospital.

Weiner reports on the value of the M.M.P.I. (Minnesota Multiphasic Personality Inventory) to predict the post-operative results of gastrectomy. He found this satisfactory, as he was able to predict the results of 87.5% of cases. Individuals with unsatisfactory operative results were those who exhibited more manifest anxiety and neuroticism on the test. I think that if this work is confirmed, it merely indicates that the M.M.P.I. can pick up the more neurotic individuals. Interviews are, as I know from personal experience, also quite an efficient means of doing the same for this purpose. It is well known that the more neurotic are the ones who do poorly after gastrectomy.

Lindman, in the article I previously mentioned, tried to find some relationship between late post-operative psychiatric symptoms and I) pre-operative anxiety, 2) sexual maladjustment, and 3) environmental factors. He found no relationship. He did find some relationship between previous depressions and the incidence of post-operative depressions. He also found that they occurred more frequently following pelvic than following abdominal operations.

In the Cincinnati study they actually found that increased anxiety and/or fear pre-operatively, was associated with the maintenance of good personality adjustment subsequently. However, a great deal of anxiety post-operatively was a poor prognostic sign for future psychiatric adjustment. Also persistent personality maladjustments were often associated with little anxiety or fear either pre-operatively or post-operatively.

In a study carried out by Doris Menzer and others in the department of psychiatry in Harvard Medical School, they were able to elucidate three factors which they thought were important in predicting reactions immediately following hysterectomy.

The first is what they call "attitude to femininity." The group who did best were those who had successfully worked through the loss of reproductive functions in the menopause. Others had denied themselves feminine gratification and turned towards masculine pursuits. These also did well. The women who did poorly post-operatively were all married and had children but "their masochism had been incompletely gratified in feminine reproductive functions." These were women whose lives revolved almost completely around the reproductive function and gratification in motherhood. A third group fell in between those two extremes.

They also found that the way a woman had handled her real and fantasied losses gave some indication of her post-operative reactions. If these had been handled by satisfactory denial and substitution, they tended to have mild to moderate reactions. The women who had little control over their feeling about losses, who had a past history of serious or recent losses of loved one and tried to handle their feelings of loss with replacement, tended to have more serious reactions. If they had lost a child, they lived with the hope of becoming pregnant again and tended to look on a hysterectomy as a permanent deprivation.

A third factor which they found important, was how the patient had previously reacted to anxiety producing situations in which there was fantasied or real threat to body integrity—previous operations, pregnancy, disease, etc. The patients who did better were the ones who were able to handle their anxieties present and past more adequately.

The Cincinnati study indicated that a functional reaction, anxiety, depression or paranoid ideas pre-operatively, was a warning in the elderly that a chronic brain syndrome might well develop. This occurred in 9 of their 11 elderly patients who developed a chronic brain syndrome post-operatively.

Summary

Some of the work being reported in the literature on the psychological aspects of surgery has been reviewed.

There are indications that a large percentage of the surgical population suffers from psychiatric illness pre-operatively. The type and incidence of immediate and late post-operative psychiatric reactions are discussed and the possible means of predicting them by psychological examination pre-operatively are indicated.

References

Bennett, A. E. and Engel: "Faulty Management of Psychiatric Syndromes Simulating Diseases." J.A.M.A., 130: 1203, 1946.

Browning, J. S. and Houseworth, J. H.: "Development of New Symptoms following Medical and Surgical Treatment of Duodenal Ulcer." Psychosomatic Medicine, 15: 328-336, July-Aug., 1953.

Deutsch, Helen: "Some Psychoanalytic Observations in Surgery." Psychosomatic Medicine, 4:105, 1942.

Dunbar, Flanders: American Journal Psychiatry, Oct., 1955, p. 277.

Elman: "Psychogenic Factors in Surgery." Surgical Clinics of North America, Oct., 1950.

Herring, F. H.: "Response During Anaesthesia and Surgery." Journal Psychosomatic Medicine, 18: 243-251, May-June, 1956.

Hebb, D. O.: Quoted by Barry Maurice, Jr. International Record of Medicine, 170: 10, Oct., 1957.

Lindemann, E.: "Observations on Psychiatric Sequelae to Surgical Operations in Women." American Journal Psychiatry, 98: 132-139, July, 1941.

Menninger, K.: "Poly Surgery and Poly Surgical Addiction." Psycho Analytical Quarterly, 3: 173, 1934.

Menzer, Doris, Morris, T., Gates, P., Sabbath, J., Robey, H., Plaut, T. and Sturgis, S. H.: "Patterns of Emotional Recovery from Hysterectomy." Journal of Psychosomatic Medicine, 19: 5, Sept.-Oct., 1957.

Sutherland, A. M., Ohrbach, C. E., Dyk, R. B., Bard, M.: "The Psychological Impact of Cancer and Cancer Surgery." Cancer, 5: 857-872, 1952.

Titchener, J., Zwerling, I., Gottschalk, L., Levine, M., Culbertson, W., Cohen, S. and Silver, H.: "Psychosis in Surgical Patients, Surg. Gynec. & Obst., 102: 59-65, Jan., 1956.
"Psychological Reactions of the Aged in Surgery." A.M.A. Archives of Neurology & Psychiatry (Cincinnati), 79, Jan., 1958.
"Consequences of Surgical Illness and Treatment." A.M.M. Archives of Neurology & Psychiatry, 77: 6, June, 1957.
Weiner, I. W.: "Psychological Factors Related to Results of Sub-total Gastrectomy." Journal of Psychosomatic Medicine, 18: 486-491, Nov.-Dec., 1956.
Welti, Walter: "Delirium with Low Serum Sodium." A.M.A. Archives of Neurology & Psychiatry, 76: 5, Nov., 1956.
Zwerling, I., Titchener, J., Gottschalk, L., Levine, M., Culbertson, W., Cohen, S., Silver, H.: "Personality Disorder and the Relationship of Emotion to Surgical Illness in 200 Surgical Patients." American Journal Psychiatry, 112: 270-277, Oct., 1955.

Some Principles for the Use of Tranquillizing Drugs

W. Donald Ross, M.D.
Associate Professor of Psychiatry, University of Cincinnati

I am glad to participate in this panel and to learn from the general discussion as well as to contribute from some of our experience in Cincinnati.

I might explain first what the nature of this experience has been. It has been in research projects carried on by the Department of Psychiatry in collaboration with the Departments of Medicine and Pharmacology. It has also been from seeing patients in psychiatric consultation where these drugs had failed or even made the patient worse as well as from the treatment of patients and the supervision of treatment of patients where these drugs were very helpful.

Rather than presenting any details of the research I shall confine myself to three general principles which arise out of this research and these consultations and treatments. These general principles appear to apply to the use of various new drugs for emotional disorders but I shall give examples to illustrate them in terms of the tranquillizers alone.

The first principle is that no drug can, by itself, solve a person's emotional problems. It may help a person to solve his problems; it may hinder him from solving them; it may even enable him to run away from solving them. The poet, A. E. Housman, said that "Malt does more than Milton can, to justify God's ways to man." The physician needs to consider whether a particular drug will help his patient solve his own emotional problems, or help the patient to use the physician's help in solving his emotional problems. If so, it will be useful. If not, then this would be an abuse of the drug. It might gain some time, but it would store up more difficulty for the future, unless the physician takes the trouble to find out what are the patient's problems or refers him to someone else who will.

Presented as a contribution to the Panel on Tranquillizing Drugs at The University of Manitoba Annual Refresher Course, April 10, 1957.

The first principle is the reason I have been unperturbed when patients have expressed the idea to me that the new drugs may do the psychiatrists out of business. I don't believe they will. Granted that I have a vested interest in the answer to this question, there are no signs that the mental health millenium is yet at hand. People still need help in solving emotional problems from physicians and psychiatrists. psychologists, case workers, ministers and lawyers, even if the new drugs can sometimes help and sometimes hinder.

The second principle is that the same drug can have different effects on different people and on the same person at different times and under different circumstances. Because of this, it is necessary for the physician to know his patient, physically and psychologically, in order to judge whether prescription of the drug would be a use or misuse.

This is a very old principle. Cicero expressed it as follows: "The confident physician makes himself acquainted, not only with the disease which he wishes to treat, but with the constitution and the spirit of the one whom he wishes to help."

The third principle is that the effect of a drug is influenced favorably or unfavorably by the patient's feelings toward the doctor and the doctor's feelings about the patient and the drug.

This principle has been demonstrated by the study of placebos and by the use of placebos for experimental controls in investigation of the new drugs. Without going into detail on the work of Beecher of the Department of Anaesthetics at the Massachusetts General Hospital, of Stewart Wolf of the Department of Medicine at Oklahoma, or of our research at Cincinnati, I will just say that placebos can alleviate or produce symptoms in more than 30% of subjects. Whether symptoms are relieved or aggravated is related to the expectations of the patient and the doctor. If the patient feels friendly to the doctor, and vice versa, and if the doctor feels that he is giving the patient something worth giving, the "placebo effect" is positive. If the converse of any of this condition is present the placebo effect is nil or deleterious. The placebo effect is produced, not only by placebos, but by active drugs. These can have added value, or less than their expected pharmacological value, depending on the doctor-patient relationship.

To use the new drugs confidently with positive placebo effect, the physician must follow the first two principles, with concern that the patient get his problems solved, if possible, and that the physician understand the constitution and the spirit of the patient.

Now for the Examples

On the first principle, that no drug can, by itself, solve a person's emotional problems:

Here is an example of the use of meprobamate (miltown or equanil):

A young man with good understanding of himself from previous psychotherapy was facing a temporary crisis. He found that he was becoming increasingly tense and over-active so that his activities were interfering with his ability to handle the crisis. He was given regular doses of meprobamate, became calm and able to apply himself to handling the crisis. At one stage before the crisis was over he stopped taking the meprobamate and had a sudden return of tension and restlessness. Later when the crisis was passed the meprobamate was decreased gradually, and there was no return of the tension.

Here is an example of the misuse of meprobamate:

A middle aged woman had suffered a death in the family and became tense and depressed. Meprobamate was prescribed with no attempt to encourage her to discuss her feelings about the deceased or to plan for readjustment of her life after the loss. The meprobamate reduced her tension for a while. She evaded facing the problems of her grief reaction. Gradually she became increasingly depressed. She was slowed up by depression plus meprobamate, not eating, losing weight, became suicidal and had to be hospitalized.

On the second principle that the same drug can have different effects on different people and on the same person at different times and under different circumstances:

Meprobamate helped a person who had muscular tension and restlessness, but it did not help a person who had feelings of inner apprehension with tachycardia and palpitation. It helped a person at a time of tension headaches, but not at a time of so-called "nervous indigestion."

Chlorpromazine helped people when they were struggling with hostile feelings and aggressions directed outwards, but it made them worse when they were reproaching themselves in a depressive turning of hostility against themselves.

A middle aged woman had had intensive psychotherapy, but her psychiatrist had left town and she saw her family doctor for symptoms related to the loss of her psychotherapist. She was given chlorpromazine and became depressed and apathetic. She improved somewhat when the chlorpromazine was stopped and resumed psychotherapy with another psychiatrist. With further improvement she contemplated a trip out of town which would be doing to her new psychotherapist what her previous therapist had done to her, i.e. leaving. She became fearful that an old phobia for travelling would return. At this time the psychiatrist advised her family doctor to prescribe chlorpromazine again, and this time it was followed by a reduction of her anxiety and of the fears in the course of the trip.

Reserpine has a different effect for a while after it has been given for about 10 days. It usually has a tranquillizing effect at first, then there is a period of increased emotional turbulence for the second week or ten days, before there is a return again to a tranquillizing effect. Some patients become frightened of the drug during this second phase and may need extra support from the physician then.

Now we come to the third principle, that the effect of a drug is influenced favorably or unfavorably by the patient's feelings toward the doctor and the doctor's feelings about the patient and the drug.

Just one example, from the misuse of meprobamate, followed by the effective use of chlorpromazine:

A young married woman was having difficulties wtih her mother-in-law. She had become tense, with headaches and difficulty in getting to sleep. She saw her doctor, ostensibly because of the headaches and insomnia, but while presenting her history she began to talk about her mother-in-law's interference in her running of her home. The doctor was busy and felt "Here is another neurotic. I hope I can settle her with equanil." He cut her short and prescribed the tranquillizer without suggesting that they discuss her problems further at another time. The patient now had anger at the doctor added to anger at the mother-in-law. She took the equanil but lay awake with a headache, ready to blow her lid.

After finding that this doctor's medicine did not help during the next week she went to another doctor who was also busy but he told her, "I understand that your headaches and insomnia are related to your feelings about your mother-in-law's interference and we should discuss this further when we can arrange the time." He gave her an appointment for a half hour undisturbed interview a few days hence and he prescribed chlorpromazine at bedtime. With this medication she slept like a baby. With a few half hour interviews over a few weeks she became able to cope with her feelings about her mother-in-law. She no longer had headaches and she slept without any medication.

These, then, are the three principles which I think you will find useful toward learning when you are using and when you are abusing the tranquillizing drugs which have been mentioned and other which are becoming available.

(1) That no drug can by itself solve a person's emotional problems. (2) That the same drug can have different effects on different people and on the same person at different times and (3) that the effect of a drug is influenced favorably or unfavorably by the patient's feelings toward the doctor and the doctor's feelings about the patient and the drug.

Pediatrics

E. Coli Diarrhoea

Fischel J. Coodin, M.D., C.M., F.A.A.P.

In recent years there has been a considerable increase in the understanding and proper management of diarrhoea in children, particularly in small infants.

The term "Epidemic diarrhoea of the newborn" was used for many years to describe outbreaks of diarrhoea, particularly in infants in nurseries. This was believed to be a clinical entity, etiology unknown. Only over the last ten to fifteen years is this concept gradually being destroyed. The situation is analagous to "Haemorrhagic disease of the newborn" which we no longer accept as a clinical entity.

In the time alloted to me, I'd like to talk about one particular cause of diarrhoea in infancy — E. coli diarrhoea. In a search for the causes of diarrhoea in infants many investigators, in past years, have looked to this organism. Escherich, after whom the organism is named, was convinced that he had found the answer to diarrhoea, before the turn of the century. In the 1920's Adam in Germany was hot on the trail. He tried to classify the many strains of E. coli according to fermentation reactions, in an attempt to isolate disease-producing strains — what he called "dyspepsie-koli." This method did not prove satisfactory. And it was not until the work of Kauffman in the early 1940's that a serological classification of E. coli was developed, which opened the door to our present knowledge.

Let's look at the antigenic structure of E. coli for a minute.

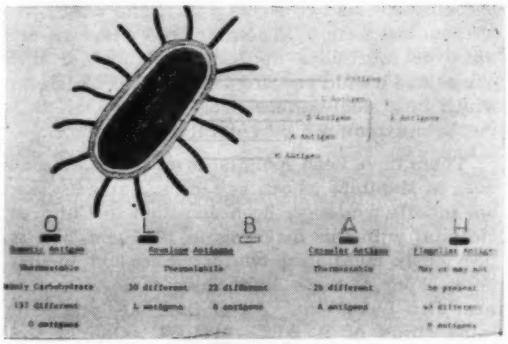


Figure 1

All strains possess an O or somatic antigen. This is thermostable, and mainly carbohydrate in composition. To date, 137 different O antigens have been distinguished. The O somatic antigen is enveloped by one of three different types of antigens termed surface, envelope, or capsular antigens, and collectively called K antigens (from the German kapseal). Each of these K antigens, termed L, A, and B, have different serological and biochemical properties. They may interfere with the action of the O antigen, but are thermolabile and can be removed by heating. The B antigens have been found in all the pathogenic strains of E. coli thus far described. E. coli strains may or may not be motile, and accordingly may or may not possess an H or flagellar antigen. So that the O and B antigens, and to a lesser extent the H antigen, are of major importance in the identification of pathogenic strains of E. coli.

Bray, in England, was the first to utilize this serological typing of E. coli to clarify the cause of an outbreak of diarrhoea. In 1945 he reported 51 infants with diarrhoea, from whose stools he isolated a serologically distinct strain, B. coli neapolitanum, subsequently termed E. coli 0111:B4. Twenty of these babies died. Since this time, epidemics and sporadic cases of E. coli diarrhoea have been reported from all parts of the world.

At the present time eleven different serotypes of E. coli are considered to be enteropathogenic (Table 1). It is expected that other types will be added to this list in the future. The types underlined, i.e. 0111:B4, 055:B5, 086:B7, and 0126:B16 have all been identified in the stools of infants with diarrhoea in Winnipeg, the last named being involved in a small epidemic which will be discussed in a few minutes.

Table 1

Esch. Coli Serotypes Associated with Infantile Diarrhoea

0111:B4	0119:B14
0 55:B5	0125:B15
0 26:B6	0126:B16
0 86:B7	0127:B 8
	0128:B10

Very briefly, human feeding experiments have confirmed the relationship between pathogenic E. coli and diarrhoea. In the United States, Neter and Shumway administered large loses of E. coli 0 111 to a two-month-old infant with multiple congenital defects, who did not harbor this organism. Within 24 hours he developed diarrhoea requiring parenteral fluids, and had large numbers of the organism in his stool. Treatment with Terramycin resulted in prompt clinical recovery and clearing of the organisms from his stool. Ferguson and co-workers used volunteers at a state prison for similar feeding experiments, and again demonstrated this relationship. The severity of the symptoms was dependent on the size of the dose administered.

Presented at the Annual Meeting of the Manitoba Medical Association, October 23, 1957.

Men who were fed non-entero-pathogenic strains of E. coli did not develop symptoms.

As to the mechanism of spread of this E. coli diarrhoea, Rogers in England has dramatically shown that it is by air contamination. Even with the best isolation techniques, the organism tends to infect neighboring infants.

What does age have to do with susceptibility to these organisms? In general, one can state that the younger the infant, the more susceptible he is to the disease. Many of the epidemics have occurred in the newborn and especially in the premature infant. It is rather infrequently found in children over the age of one year. However, adults, especially elderly debilitated patients, have been found to have diarrhoea due to these organisms.

What happens to the organisms in the stool? Following spontaneous recovery they may be excreted for days, weeks, or months — a true carrier state. Treatment with neomycin or another antibiotic will result in rapid clearing of the organisms from the stool, but they will soon reappear, even in the continued presence of the antibiotic, with or without a recurrence of the diarrhoea. A recent study in England indicates that as many as 25% of babies in nurseries are carriers of pathogenic E. coli, and 1 - 2 percent of the general population are, similarly, carriers.

Blood antibody studies for these enteropathogenic E. coli have been only partially successful, and will not be gone into at this time.

Now, in Winnipeg, two babies were admitted to the Children's Hospital with diarrhoea on Dec. 12, 1956. One of these was dead on arrival. He had been born at another hospital, and had remained there for approximately three months awaiting adoption. During this time he had had an upper respiratory infection and a mild diarrhoea. He was placed in a home one week prior to his death, and during this week he had had a moderate degree of diarrhoea. On the morning of his death, this infant had several rather explosive diarrhoeal bowel movements. Autopsy cause of death was found to be infantile diarrhoea due to E. coli 0126:B16, with severe dehydration.

On the same date, another infant, one and one-half months old, was transferred to Children's Hospital from the same hospital, where he had been hospitalized for almost a month with an upper respiratory infection, poor feeding, failure to gain weight, and diarrhoea. The baby was emaciated but not grossly dehydrated. Culture of his stool grew E. coli 0126:B16 — the same strain. He was treated with neomycin orally and supportive measures, and was discharged well after two and one-half weeks.

I may mention here that the signs and symptoms of E. coli diarrhoea are indistinguishable from infantile diarrhoea of other causes. The stool has no particular odour or appearance, and the blood count is of no particular help. In fact, even at autopsy there are no specific findings. This is a purely bacteriological diagnosis.

Some three or four days after the admission of these two patients, there was a sudden outbreak of diarrhoea among the babies in the nurseries of the previously-mentioned hospital. On Table 2

Table 2

Case	Birth Date	Diarrhoea	Type of Stool	Weight		Stool Culture
A	14-11-56	Dec.25-31 1.1.1.1.2.1.0.	Loose, yellow	8-4	8-14	E.Coli 0126
B	13-11-56	Dec.27-31 1.1.1.2.0.	Loose,yellow,curdly	9-13	9-9	No pathogens
C	3-12-56	Dec.25-Jan.1 3.1.1.1.1.1.0. Jan.24 4.	Loose,yellow,curdly Loose,yellow	8-3 8-14	8-3 9-2	No pathogens No pathogens
D	5-12-56	Dec.28-Jan.1 7.1.4.0.0. Jan.21-26 2.0.1.1.0.1.	Watery, greenish-yellow Watery, greenish-yellow	6-14 10-4	9-3 10-3	No pathogens No pathogens
E	30-8-56	Dec.27-Jan.1 1.2.3.3.3.2.1.	Watery,curdly,gr-yellow	6-3	6-9	No pathogens
F	6-12-56	Dec.27-Jan.1 1.7.3.3.1.1. Jan.20-24 1.0.3.0.4.	Watery,curdly,gr-yellow Watery,curdly,yellow	6-13 8-3	7-1 8-5	E.Coli 0126 No pathogens
G	24-11-56	Dec.26-30 6.4.4.3.2.	Loose,chalky,yellow	6-2	6-4	E.Coli 0126
H	7-10-56	Dec.26-30 8.4.3.0.0.	Watery,yellow	9-4	9-13	E.Coli 0126
I	28-10-56	Dec.26-30 4.6.3.3.2.	Loose,greenish-yellow	8-4	8-4	E.Coli 0126
J	31-10-56	Dec.26-Jan.1 3.3.3.3.1.0.	Loose,watery,yellow	6-12	6-9	No pathogens
K	4-10-56	Dec.26-27 1.2.	Large,foul,yellow	7-12	7-12	E.Coli 0126

you can see that eleven babies were involved. Cultures showed the presence of E. coli 0126:B16 in the stools of six of them, and it is a reasonable presumption that the rest were of the same etiology. All the babies in the nurseries were promptly started on neomycin orally, and perhaps for this reason the epidemic proved to be very mild. As you can see, the babies ranged from three weeks to four months of age; many of them had only one or two stools a day, and only a few of them lost weight. The diarrhoea averaged only three or four days in duration, although three babies had a mild recurrence three weeks later, at which time stool cultures were negative. None of the babies required parenteral fluids.

There have been sporadic cases of E. coli diarrhoea in Manitoba as can be seen in Table 3. These are infants who have been admitted to Children's Hospital with this diagnosis in 1956 and '57. The previously mentioned two cases are not included here. Four of these eight cases came from in or near Red Lake, Ont. All of them are due to the same strain of E. coli — 0111:B4. One of them had a mild recurrence of his diarrhoea two weeks later, with negative stool cultures. One infant came from West Selkirk, and three from Winnipeg. They ranged from two weeks to eighteen months of age. Almost all of them received neomycin, along with whatever other measures were deemed necessary. They all survived and were discharged well. The last case was in hospital only last week

Table 3

Name	Admission Date	Age	Home	Symptoms	Type of E. coli	Treatment
.J.	June/56	3 wks.	Red Lake Ont.	v. & d. for 1 wk.	0111:B4	neomycin, i.v. fluids supportive
.J.	July/56	5 wks.	"	recurrent diarrhea	no E. coli found	dietary only
.D.M.	June/56	10 1/2 mo.	Red Lake Area	U.R.I. & diarrhea for 1 mo	0111:B4	neomycin & diet.
.C.M.	July/57	17 mo.	W.Selkirk Man.	diarrhea for 13 d.	0111:B4	neomycin
.M.	Aug./57	7 mo	Red Lake Area	diarrhea & vomiting	0111:B4	neomycin, chloram., i.v., & support.
.L.	Sept./57	4 mo	Wpg.	diarrhea for 1 wk.	0111:B4	neo, chloro., terra, sulpha
.B.	Nov./56	2 mo	Red Lake Area	v. & d. for 1 wk	0111:B4	neo, chloro., i.v.
.W.	Nov./56	18 mo	Wpg.	diarrhea 1 1/2 wks.	055:B5	diet
.M.	Oct./57	2 wks	Wpg.	diarrhea	086:B7	chloro.

with a strain of E. coli — 086:B7 — which is new for this area.

To give you an idea of the proportion of infantile diarrhoea due to enteropathogenic E. coli, the experiences of the Children's Hospital of Washington, D.C., are listed in Table 4. Of a total of 957 stool cultures of children with diarrhoea, they found a Salmonella in 4,2 percent, a Shigella in 7.0 percent, and pathogenic E. coli in 12.1 percent of the cases.

Table 4

Total Number of Stool Cultures: 957

ISOLATION OF:		
SALMONELLA SP.	40	4.2%
SHIGELLA SP.	67	7.0%
E: COLI:		
0127	98	
0111	8	
055	5	12.1%
026	4	

In table 5 you will see the experiences at the St. Louis Children's Hospital in 1955. Of 123 admissions with acute diarrhoea, they demonstrated the presence of pathogenic E. coli in the stools of fifty. Only one infant died, and this was due to a complicating staphylococcal sepsis. The various strains of E. coli are listed on the right; on the left are the other organisms grown from the stools. Notice 25 cases where non-pathogenic E. coli were isolated; five cases of Salmonella infection, and six cases of Shigella. The incidence of E. coli diarrhoea would seem to vary markedly from year to year. In 1956 they had only about twenty admissions with E. coli diarrhoea at the same St. Louis hospital, compared with fifty in eleven months of 1955.

In the treatment of this type of diarrhoea, the broad-spectrum antibiotics are generally effective, but the drug of choice is neomycin. It should be used orally in a dose of 20 mgm. per pound body weight per day. The well-established principles of treatment of diarrhoea, including intravenous fluids and diet, are as essential to treatment as the antibiotics.

Table 5

Stool Cultures in 123 Infants with Acute Diarrhoea Admitted to the St. Louis Children's Hospital, Jan. 1 to Nov. 1, 1955

ORGANISM	PATIENTS	TYPE OF PATHOGENIC *Esch. coli* ISOLATED	
Pathogenic *Esch. coli*	50	*Esch. coli* 0127:B 8	41
Proteus strains	29	0111:B 4	3
Aerobacter aerogenes	27	0 55:B 5	2
Nonpathogenic Esch. coli	25	0125:B15	2
Pseudomonas aeruginosa	11	0126:B16	2
Paracolon	7	0 86:B 7	1
Shigella	6	0 26:B 6	1
Salmonella	5	0112a, 112b:B13	1
			53

(3 patients had 2 different strains in their stools)

Total Patients	123
Diarrhea due to pathogenic *Esch. coli*	50
Deaths (due to staphylococcic sepsis)	1

Summary

1. Certain serotypes of E. coli can produce diarrhoea in infants. 2. In an attempt to establish the etiology of an infant's diarrhoea, it is essential to perform routine serotyping of the E. coli isolated from the infant's stool. This is a simple procedure in any routine bacteriology laboratory. 3. E. coli diarrhoea may spread rapidly through hospital wards despite the use of modern isolation techniques. 4. Patients with E. coli diarrhoea cannot be distinguished on clinical grounds from patients with non-specific diarrhoea. 5. The treatment of patients with E. coli diarrhoea consists of the employment of the well-established principles of treatment of infantile diarrhoea plus oral neomycin. 6. The experience with E. coli diarrhoea in Winnipeg have been presented.

Acknowledgment

Without the invaluable help of Dr. Ralph Robertson the epidemic experiences could not have been catalogued and reported.

Selected References

1. Levin, S.: J. Pediatrics, 52: 227, 1958.
2. Gamble, D. R. and Rowson, K. E. K.: Lancet, 619, Sept. 28, 1957.
3. Quilligan, J. J. et al: A.M.A. Am. J. Dis. Child., 93: 420, 1957.
4. Quilligan, J. J. et al: A.M.A. Am. J. Dis. Child., 95: 134, 1958.
5. Cooper, M. L. et al: Annals N.Y. Acad. Sciences, 66: 78, 1956.
6. Ferguson, W. W.: Annals N.Y. Acad. Sciences, 66: 71, 1956.
7. Brown, E. H. and Bradley, E. H.: Lancet, 1218, Dec. 14, 1957.
8. Whitaker, J. et al: Am. J. Dis. Child, 95: 1, 1958
9. Stulberg C. S. et al: Am. J. Dis. Child., 95: 30, 1958.
10. Wheeler, W. E. and Wainerman, B.: Pediatrics, 14: 357, 1954.

Editorial

S. Velsrub, M.D., M.R.C.P. (Lond.), F.R.C.P. (C.), F.A.C.P., Editor

Medical Choreography

Were it not for the gay young creature flitting about ecstatically from partner to partner in the course of a lively Paul Jones at the Annual Dance, the ensuing lines would not have been written. Nor would your limping editor, drawn into the whirlpool much against his better judgment (this dance is notoriously unkind to lumbar discs) have had the occasion to reflect upon the dance, and stray from the straight and narrow paths of Aesculapius to the twisting bends and turns of Terpsichore.

Why is the Paul Jones so popular and so enduring? The answer may well be in the realm of the subconscious, where the hidden wishes play on the road to symbolic expression. The Paul Jones gives the expression to at least three basic wishes. The first and most obvious is the wish for a mate. The second, not quite to obvious, but no less real, is the desire to change partners, the subconscious rebellion against monogamy. The third is the wish to belong, to be one in a crowd, to join hands in a big circle. The dance, like the dream, is the fulfilment of subconscious drives.

The symbolism of the dance is a favourite subject with the psychoanalysts. The religious dances of primitive tribes, the dances of the Vestals in the temples of ancient Rome, the savage rhythms of the Indian Sun Dance, as well as the therapeutic medical dances—the dances of the witch doctor and his entourage in the ritual of exorcism, have been for years providing grist for the mills of analysis. It is perhaps a pity that modern civilized medicine finds no therapeutic uses for the dance. Alas! not even the most "up to date" hospital, which provides all the comforts of a resort hotel, has the courage of supplying dancing girls as aids to therapy.

If not in the field of therapy, has the dance any applicability in other fields of medicine? Apparently none, unless one attaches importance to such descriptive terms as "hilar" dance, "dance of the arteries," "dance de ventre" (accelerated peristalsis) "dancing mania," "St. Vitus' dance"—an importance hardly warranted by their trivial function.

Yet, it would seem that with a little imagination spiced with frivolity one should be able to find a measure of usefulness for the "light fantastic" under the heavy skies of medicine. Take, for instance, the field of medical teaching. Could not the dance illustrate the rhythmic functions of the body as effectively as the medical drawing has portrayed its structure? Dancing teams of delicate auricles and muscular ventricles could bring home to the student the cardiac rhythms and its disturbances as no verbal description would. A good "pulmonary" adagio dancer could illustrate "Cheyne - Stokes" while a "cerebral" flamenco expert throws a magnificent fit. The possibilities are unlimited.

The idea unfortunately has not caught on. No medical college to date has added an accomplished ballerina to its teaching staff. Yet, there are straws in the wind. In his book on "Fluid and Electrolytes in Practice," Harry Statland explains the milliequivalent system as follows: "One might liken this to the hostess making up her list of guests to a dance. She does not invite 1000 pounds of girls for 1000 pounds of boys. Rather she is interested in how many of each and regardless of differences in weights, the number of males and females (anions and cations) must be equal."

When a respectable medical writer visualizes anions and cations as male and female dancers, he leaves the door open for others to expand the vision and view the whole electrolyte structure as a gigantic underwater ballet, a kind of aquacade with male (tom) cations and female anions dancing joyously in the salty brine of the milieu interieur. The music, of course, would be provided by the famous endocrine orchestra with the pituitary as its acknowledged leader.

An imaginative choreographer, one able to create dances for every rhythmic function of the body and every dysrhythmia, could become as important to medical teaching tomorrow, as a medical illustrator is today. He could easily become indispensable to every medical college in the land.

Should he find the financial rewards of academic pursuits depressingly meager, our medical choreographer could readily supplement his income by "part-time" services to various medical institutions. He could lend his talents to the staging of a "Valse Triste" for our Public Relations, "song and dance" for our politicians, and—to end on a cheerful note—"The Anniversary Waltz" for our joyful anniversaries.

Ed.

Book Review

Heroic and Bitter End

Death of a Man: Lael Tucker Wertenbaker. 180 pp., Toronto, Random House.

Death and the process of dying make grim subjects for a story, especially when the story is a true one. It may well be that a major portion of the great literature of the world deals with death. Shakespeare litters his stages with the dead, and in nearly all of the classical tragedies someone sooner or later is destroyed. But in most of these, death is a climactic event, sudden in its occurrence and necessary as the only tidy and logical solution to a tragedy.

In "Death of a Man" on the other hand, death is the main theme; the painful process of dying as it affected a man and involved his wife is the whole story.

Charles Wertenbaker, author and one time foreign editor for Time magazine, developed cancer of the bowel while residing in France. With his wife, the author of the book, he travelled back to New York to have an operation performed by the only surgeon he trusted. The operation revealed that the tumor could not be removed. He then returned to France for the few remaining months left to him and there he died after a series of attempted suicides, the final one succeeding only with the active help of his wife. It's as grim as that and no book for a weak stomach!

And yet the universality of death makes this a story that should be told and that ought to be heard. It is trite but only too true to say that death is an experience that sooner or later all of us will have; but emotional reaction, once we have bought our life insurance, forces us to turn away from any consideration of that terminal event. This story focuses our attention on death and holds it there for the duration of the narrative.

With all this, the story by Lael Tucker Wertenbaker is beautifully written, with kindness and deep understanding; at times it reaches heights of great prose. It could have been so written only by a gifted person who experienced the agony and who felt the pain of a loved one about to die.

This Way?

Any man who dies in this epic manner is by definition a hero. And it may offend canons of good taste to be critical of a heroic character now dead. But should it not be asked: Was all the heartache and misery really inevitable? Could not some of the doleful and painful incidents have been avoided? The answer from a clinical and humane point of view is that a good deal of the suffering undergone by Charles Wertenbaker and much of the consequent misery to which his understanding and compassionate wife was subject, was tragically unreasonable, unnecessary and avoidable.

Charles Wertenbaker was a man obviously accustomed to making decisions — final decisions — for himself and for others during his active lifetime, right to the bitter end. "It was a principle with him . . . that he had a right to die as he wished to, when he chose, if possible." He was equally determined to make all the decisions throughout his illness down to the termination of his life. At this unhappy and critical period, for his family as well as for himself, it was his determination to live with "dignity" for as long as this was possible and then to end his life.

"Dignity" in this context is difficult, indeed impossible, to define. Charles Wertenbaker's definition of dignity was to go through the process of dying with a minimum of outside interference. From his symptoms he suspected the presence of his disease for over a year before he took the proper steps resulting in a diagnosis. By this time the disease was hopelessly advanced. When the decision for surgery was made Wertenbaker dictated in detail the conditions which were to determine for the surgeon the extent of the operation. This reduced the value of a surgeon to the function of a technician.

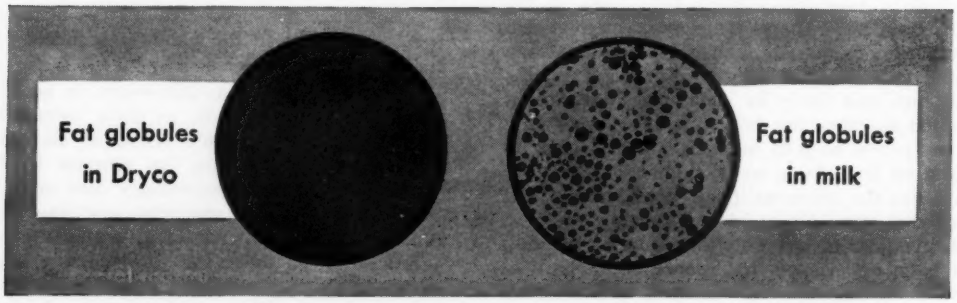
After the operation, this desire to go it alone, admirable as it may be in some circumstances, resulted in an avoidable series of embarrassing misadventures while on the journey to France. There followed an unnecessary and complicated business of smuggling morphine and other essential sedatives, on an international scale. During the terminal period there was an excessive degree of suffering which a relatively minor operation could have relieved. This was refused. And finally there were several attempts at suicide, the last one being effective only by the help of his wife. Truly a tragic and needlessly involved procedure.

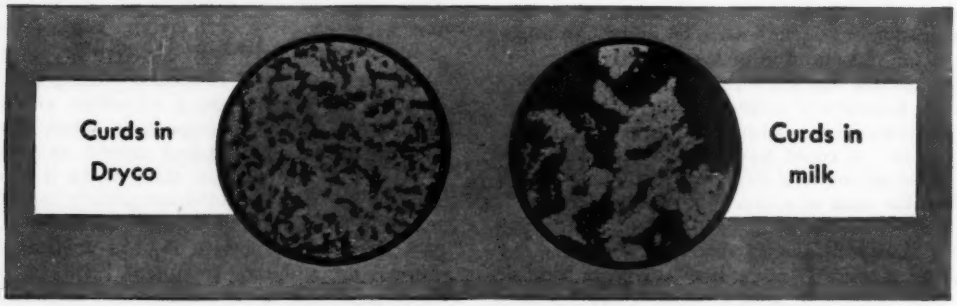
It seems a pretty elementary thing that if one sets out to do something of which one has no experience whatever (especially when the thing may be as complicated and painful as a terminal illness) the only sensible thing to do is to consult experts and to be guided completely by their advice. In this sphere doctors are the experts. There is a large and important field in medical therapy known as palliative treatment. This is particularly applicable to the treatment of terminal cancer and includes the uses of sedatives in whatever amount or variety becomes necessary. It also includes certain types of surgical operations, which, while they may not increase life expectancy, do prevent some of the painful complications that sometimes occur.

Charles Wertenbaker, because he chose to die "with dignity" chose to deny himself this help. His right to do this to himself is not in question.

DRYCO–ideal formula for normal infants as well as prematures

1. **High Protein-Low Fat Ratio:** 2.7 to 1 ratio of protein to fat facilitates preparation of *low-fat* formulas which assure *ample protein intake*—to meet the normal growth requirements of early months in normal infants *and* the tissue-building diet requirements of premature and malnourished infants.
2. **Carbohydrate Flexibility:** Dryco's moderate lactose content permits flexible regulation of carbohydrates at the doctor's discretion. Dryco may be used: alone; with added carbohydrate; with added whole milk; or with added carbohydrate and whole milk.
3. **Extreme Digestibility:** Fat globules and protein curds in Dryco are more digestible.





DRYCO

For further information write: **THE BORDEN COMPANY LIMITED,**
Formula Foods Department, Spadina Crescent, Toronto.

But the torment and suffering of the woman who loved and cared for him during all this trying time, was more than one person should inflict on another, no matter what the circumstances.

There is a danger, because this book is so well written and the central characters so heroic, that the impression may be conveyed that this is the way to die with cancer. Nothing could be further from the truth. It would be tragic indeed if Charles Wertenbaker's example were followed by even a single person. This is no way for any man to die.

A. A. K.

—Reprinted from Winnipeg Free Press, Apr. 26, '58.

♦

Obituaries

Dr. William Jesse Grant

Dr. William Jesse Grant, 89, died on July 17. Born at Richmond Hill, Ontario, he was educated there and at Toronto University before coming west in 1892. He taught at various schools in Manitoba before entering Manitoba Medical College from which he graduated in 1905. He practised at Norway House, Manitoba, and at other posts in Manitoba and Saskatchewan, returning to Winnipeg in 1929. During the first World War he served in the R.C.A.M.C. with the rank of captain. He is survived by two daughters.

♦

Dr. Magnus Hjaltason

Dr. Magnus Hjaltason, 84, died at his home in Winnipeg on August 29th. Born in Iceland, he came to Manitoba 67 years ago and graduated in 1909 from Manitoba Medical College. He practised at Glenboro and Lundar before retiring to Winnipeg ten years ago. He is survived by his wife, four daughters and one son.

♦

Dr. Edward Thomas Etsell

Dr. Edward Thomas Etsell, 65, died on August 24th in Misericordia Hospital. Born in Virden, he was educated there and in Winnipeg, receiving his Arts degree in 1915 and his M.D. in 1920 from the University of Manitoba. In 1936-37 he studied urology in London, Paris and Berlin and served for a year in the department of urology, Hotel Dieu, Montreal. He spoke French fluently, had some knowledge of German, Italian and Ukrainian, and rarely missed a symphony concert. He is survived by his widow.

Medical History

Samuel Willis Prowse, M.D.
1869 - 1931
Fourth Dean of Medicine

Ross Mitchell, M.D.

A generation of medical students who know of Dean Prowse only by name has passed through medical college halls and gone out into the world. It seems fitting, therefore, to set down in writing something of the Dean who guided Manitoba's medical school through the transition from almost wholly didactic and clinical teaching to a course in which clinics are combined with experimental and laboratory work. In this change Doctor Prowse was well qualified to direct and his efforts were crowned with success.

He was born at Murray Harbour, Prince Edward Island, August 25, 1869. His grandfather William, had come from Devonshire to the Island in 1823, and his father Samuel was a successful merchant who interested himself in provincial politics and later was appointed to the Senate of Canada. The boy was happy in his parents and in the place of his birth. Of the latter, Bruce Hutchison writes:

"No traveller, however insensitive, can fail to see, as soon as he has landed, that The Island is not merely a speck of soil detached from the mainland by water, but a state of mind, detached by a much wider word of thought."

So The Island gave the boy independence and equanimity and his parents were able to see that he had a good education. He took his arts course at Mount Allison College in Sackville, New Brunswick, 1885-1889, then went to Edinburgh, whose medical faculty ranked high. He took the M.B., Ch.B. degree in 1896 and the F.R.C.S. (Ed.) in 1898. From 1894-1898 he assisted and practised at Colinsburg, Fifeshire.

In 1898 he came to Winnipeg, then in a period of expansion, and opened an office on Main Street. In 1902 he was appointed lecturer in physiology in the old brick college at Kate and McDermot. Previously the teaching of physiology had been purely didactic but he got together a kymograph and other apparatus for frog muscle-nerve demonstrations, pressed into service one of his students, had large scale drawings made of the ear and other organs and lectured on the structure and function of muscles and nerves to make his students realize something of the magic of life.

After a year or two of general practice he entered the special field of ophthalmology and otolaryngology in succession to Dr. J. W. Good, the second Dean of the college.

In 1910 he was president of the Winnipeg Medical Chirurgical Society, soon to link with the Clinical Society to form the Winnipeg Medical Society.

In August 1914 the first World War broke out. Manitoba quickly sent two or three Field Ambulances and a Casualty Clearing Station. By 1916 Canada had three divisions in the Field and a fourth was being readied for the Somme offensive. It was decided that Manitoba Medical College should raise a Casualty Clearing Station and Dr. Prowse was appointed Commanding Officer with the rank of Lieutenant-Colonel. The ranks were speedily filled and in June 1916 the unit marched from the Medical College to the C.P.R. Station. There were high hopes that the C.C.S. would accompany the Fourth Canadian Division to France, but on arrival at Bramshott Camp, the unit was split into groups and assigned to various hospitals. It was characteristic of Colonel Prowse that he insisted the unit would again be one. With a devoted orderly he kept headquarters in a tent on Sir John Moore's plains throughout a bitterly cold winter, an experience which probably undermined his health. He was right in his belief for the unit was later regrouped and sent to France.

In 1917 Dr. H. H. Chown, the third Dean and one who had done much for medical education, brought Manitoba Medical College more closely into the orbit of the University. The new medical building on the present site, opened in 1906, and its equipment all free of debt was handed over to the University of Manitoba and its faculty became the Faculty of Medicine. Dr. Prowse was summoned from France to become the fourth Dean on the resignation of Dr. Chown.

This move was part of a great forward step in medical education. Early in the century the American Medical Association became keenly interested in better medical teaching. The Carnegie Foundation for the Advancement of Teaching was approached and it consented to grant funds and workers to investigate the state of medical education in America. The Flexner report of 1910 to the Carnegie Foundation was revolutionary. Its purpose was to establish and enforce minimum standards for medical schools. The report showed that there were 160 medical schools in the United States. Some were excellent, others were diploma mills. Within a relatively short time the number of medical schools was reduced by more than fifty percent. Medical schools were graded in six classes A to F. The report recommended that Class F schools be encouraged to raise standards and equipment. The University of Manitoba accepted the recommendation.

In 1921, with the aid of funds from the Rockefeller Foundation, a building was erected for physiology, biochemistry and bacteriology and in 1922 an addition was made to the main building for pathology and Dr. Gordon Bell's provincial

laboratory. In 1942 our school was graded Class A and, to commemorate this, a dinner was held in the Fort Garry hotel at which Dr. Prowse gave a notable address on medical education. The Faculty of Medicine, members of the "Old Faculty," the Medical Alumni Association and the College of Physicians and Surgeons of Manitoba set up the Prowse Prize for original research. The Prize is a bronze medal and $250.00 in cash. The names of the holders are an index of the stimulus the Prize has given to research: H. D. Kitchen, Herbert Meltzer, Harry Medovy, C. H. A. Walton, B. M. Unkauf, S. D. Schultz, M. D. Campbell, L. R. Coke, Sara Dubo, D. W. Penner, R. E. Beamish, J. B. R. Cosgrove, R. M. Cherniack, L. J. Cera, W. Lingg, R. W. Irwin, D. P. Snidal, A. W. Krisman and B. J. Kaufman. During the Prowse deanship the course leading to the B.Sc., Med. degree was set up.

When the British and Canadian Medical Associations met jointly at Winnipeg in 1930, Dr. Prowse received from the University of Manitoba the LL.D. degree, honoris causa, in company with Dr. Harvey Smith, President of both Associations, Mr. Burgess of Manchester, past president of the B.M.A., Lord Moynihan of Leeds, the Lister orator, Lord Dawson of Penn, King George V's physician, and other distinguished guests.

Dr. Prowse's health was never robust and on August 1, 1931, he died of coronary thrombosis. The President of the University, Dr. J. A. MacLean said of him: "the memory of his kindness will outlive even the record of his achievements." This was a fitting tribute to a man who had high qualities of mind and of heart.

Impressions of the 2nd World Congress International Federation of Gynaecology and Obstetrics, Montreal June 22 - 28, 1958

Ross Mitchell, M.D.

It is evident that Canada has come of age in the scientific medical world when she can play hostess to a gathering of the representatives of 58 nations and conduct a program lasting for a week. The congress was held in the new and lavish Queen Elizabeth Hotel. Arrangements for the meeting were carried out by a hard-working group of Montreal doctors headed by Dr. Leon Gerin Lajoie, President of the Federation. A word of praise must go out to the Ladies' Committee whose help was invaluable.

The opening ceremony was full of colour. After the platform party was seated, R.C.M.P. constables in scarlet headed a procession of cadets in khaki bearing of the flags of the nations represented, mounted the platform and stood stiffly at attention at the rear. Some ten minutes later the flag-bearer of Israel, who had been so anxious over his assignment that he could not eat dinner, thudded in a dead faint and had to be carried back-stage. The introductory remarks were mercifully brief save when a federal minister read a speech in English of thirty-five minutes duration.

The official languages for the Congress were French, English, German and Spanish. Headphones were available and translations were into English and French. One of the Russians spoke in his native tongue but he had thoughtfully provided the interpreter with a German text which was rendered into French and English. On each of the five days there were main lectures on general topics, free communications on obstetrics in one room, on gynaecology in another, with scientific films shown in a third room. There were also round table discussions which brought together the elite in their particular spheres. Encircling these rooms were the scientific and commercial exhibits which well repaid a visit. In all the divisions, Manitoba was well represented.

The Mayor of Montreal, M. Sarto Fournier, received the guests at a civic reception in the chalet on top of Mount Royal on Monday evening. The delegates mingled freely to become acquainted and another opportunity for making friends was afforded by the dinner on Wednesday evening. There was a tour to the St. Lawrence Seaway, another to Laurentian resorts, a visit to the Botanical Gardens and a fur show.

After the impressions of numbers and of colour, fun and froth, come sober reflections like those of little Peterkin "but what good came of it at last?" The judgment is that the meeting was well worth while. Gathered together were men and women from all corners of the earth, China, Japan, Indonesia, Iran, Yugoslavia, Nigeria, Egypt, Cuba, Uruguay, differing in race, creed, language and dress but drawn together by a common devotion to science. In one round table conference the moderator came from Okayama, Japan, the members from France, Austria, India, United States, Argentine, England, Italy and Russia. Present were internationally known figures such as Kottmeier of Stockholm, Brunschwig of New York, Peel of London, Jeffcoate of Liverpool, Antoine of Vienna, Selye of Montreal and de Watteville of Geneva. It was a humbling experience to hear Professor Caldeyro-Barlia of Montevideo deliver an address, crisp, rounded and well illustrated, in perfect English. As so often happens the top men in their fields were the most accessible. Living through an experience such as this 2nd World Congress afforded makes one think that Robert Louis Stevenson's tribute to medical men was not unfounded.

TAKE A NEW LOOK AT ALLERGENS*
TAKE A LOOK AT NEW DĪMETANE

There is no antihistamine better than DIMETANE for allergic protection. DIMETANE gives you good reasons to re-examine the antihistamine you are now using: unexcelled potency, unsurpassed therapeutic index and relative safety...minimum drowsiness or other side effects. Has been effective where other antihistamines have failed. DIMETANE Extentabs® (12 mg.) protect for 10-12 hours on one tablet. Also available: Tablets (4 mg.), Elixir (2 mg. per 5 cc.).

A. H. ROBINS CO. OF CANADA, LTD., MONTREAL
Ethical Pharmaceuticals of Merit Since 1878

*Typical Allergens: Animal Hair and Dander • Pollen • Molds • Bacteria and Viruses • Feathers • Insect Scales • Vegetable Fibers and Seeds Plant Juices • House Dust • Drugs and Chemicals • Minerals and Metals.

Robins

Dīmetane® (PARABROMDYLAMINE MALEATE)

Social News

Reported by K. Borthwick-Leslie, M.D.

The annual meeting of the College of General Practice, Manitoba Chapter, at Clear Lake was a great success.

The weatherman was not too kind, and from reports the Chalet management not too cooperative as to finances, but the scientific meetings and displays were all excellent, interesting, informative and practical.

The gambling spirit of the local members seems to be slipping, however, I couldn't find **one** soul willing to take a chance on me and my Karmann Ghia "bug" for the trip out. Could be the package deal accomodation discouraged 'em!

♦

We bid a fond farewell to Tom Casey, who left last week for his post-graduate work in London, England.

Tom will be sadly missed both by all his friends in the profession, and the multitudinous activities in sport, children's guidance classes, etc.

♦

Dr. and Mrs. Joseph Ward now are much more imbued with the spirit of adventure. Their cruise via the Red River in their houseboat "The Little Ark" to Grand Forks, must have been a lot of fun.

♦

Dr. J. W. Whiteford has been elected president of the Men's Musical Club. Dr. Whiteford succeeds Reg Hugo, who has held the post for some years.

♦

The Winnipeg Clinic welcomes to its staff Kenneth C. Finkel, B.A., M.B., M.R.C.P., D.C.H. Dept. of Paediatrics; William May, M.B., B.S. (London), L.R.C.P., M.R.C.S., D.O. (R.C.S.) in the Dept. of Ophthalmology; and Frederick P. Waugh, B.Sc., M.D. to the Dept. of Internal Medicine.

♦

The Manitoba Clinic announces that Sheldon G. Sheps, M.D., B.Sc. (Med.) recently of the Department of Medicine, Beth Israel Hospital and Harvard Medical School, Boston, Mass., and the section of Vascular Disease, Mayo Clinic, has joined their Department of Medicine and Cardiology.

♦

Miss Margaret Hillsman, daughter of Dr. and Mrs. John A. Hillsman, having spent a year on the continent, is doing post graduate work at Radcliffe-Harvard school of business administration, Boston.

♦

Cupid has been particularly accurate aiming his darts at our profession:

♦

June 25, 1958, Lila Diane, daughter of Drs. Bella and Louis Kobrinsky, became the bride of Harvey Karnberg. The young couple have made Calgary their home town.

♦

August 10, 1958, Sheila Ann, daughter of Dr. and Mrs. M. M. Pierce was married to Dr. David Martin Brodovsky, son of Mrs. N. S. Brodovsky and the late Mr. Brodovsky. They are in residence at the Medical Centre, Winnipeg.

August 9, 1958, Anne Marie, daughter of Mr. and Mrs. T. B. McConnell, and Dr. Myles MacLennan, son of Mrs. Angus MacLennan and the late Mr. MacLennan, Regina, Sask., plighted their troth in St. Ignatius Church. Dr. and Mrs. MacLennan will reside in Regina.

♦

September 13, 1958, Westminster Church was the scene of the marriage of Carol Anne, daughter of Dr. and Mrs. Francis Hartley Smith to Dr. Richard Kent Macoomb, son of Mr. and Mrs. Robt. S. Macoomb, Fort Garry.

♦

August 16, 1958, at Melita, Man., Beverley Ann Murray became the bride of Dr. Valdimar B. Kjernsted of Ashern, Man.

♦

August 22, 1958, Jean Edith, daughter of Dr. and Mrs. George W. McNeill of Stony Mountain, became the bride of Dr. Lawrence Edward Matrick, son of Mr. and Mrs. Matrick, St. Mary's Road, St. Vital, Man.

♦

September 18, 1958, Arlene Meredith, daughter of Dr. and Mrs. Edward Johnson, Selkirk, Man., will be married in Westminster Church to William T. Bedwell, son of Mrs. Robert L. Bedwell and the late Mr. Bedwell.

♦

September 20, 1958, Nancy Ann, daughter of Dr. and Mrs. T. Edward Holland will become the bride of Roy Hamilton Parkhill — St. Andrew's River Heights United Church, 3.30 p.m. the time and place.

♦

September 20, 1958, in St. Stephen's Anglican Church, Bevis Doreen Francis will become the bride of Dr. Richard Roland Bird, son of Dr. and Mrs. Roland Bird.

♦

Welcome to our newest arrivals, and congratulations to the happy parents:

Dr. and Mrs. B. Derbach announce the arrival of a daughter, August 28, 1958.

♦

Dr. and Mrs. R. F. Burns, Rochester, Minn., announce the birth of Graham Bruce, Sept. 6, 1958, baby brother for Catharine and Tannis.

♦

Dr. and Mrs. R. G. Handford welcome Lorraine Ruth — August 15, 1958 — small sister for Jamie.

♦

Dr. and Mrs. Dallas Medd announce the birth of a sister for Lorna, Kenneth and Kathryn.

♦

Dr. and Mrs. Noel Joubert (nee Dr. Doreen Papageorgiou) welcome their son, Richard Noel, in Brandon, Man., July 21, 1958. (Sorry, I must have missed you previously, Doreen).

♦

Dr. and Mrs. Keith Stinson, Flin Flon, Manitoba, announce the arrival of David Keith, September 11, 1958.

Detailmen's Directory

Representing Review Advertisers in this issue, whose names are not listed under a business address.

Ames Company of Canada, Ltd.
W. H. Davis ..... VE 2-4869

Arlington-Funk Laboratories, division U.S. Vitamin Corp. of Canada, Ltd.
Ed Lessor ..... GL 3-0807

Ayerst McKenna and Harrison
W. R. Card ..... HU 9-7115
C. C. Savage ..... SU 3-4558
Jack Ostrow ..... ED 4-3240

Borden Company Ltd.
Ken Hodges ..... AL 3-4319

British Drug Houses
W. S. Langdon ..... GL 3-1325
H. Harvey ..... TU 8-5341

Calmic Limited
Ken Harrison ..... VE 2-4120

Ciba Company Ltd.
Edward Stephany ..... HU 9-7292
Leslie D. MacLean ..... CE 3-3240

Connaught Laboratories
Brathwaites Ltd. ..... WH 2-2635

Cow & Gate (Canada) Ltd.
Keith Searle ..... ED 9-1347

Frosst, Charles E.
W. M. Lougheed ..... HU 9-3963
W. J. McGurran ..... AL 3-0722
E. R. Mitchell ..... HU 9-6164
R. P. Roberts ..... AL 3-4032

Geigy Pharmaceuticals
L. H. Lockshin ..... JU 9-5472

Hoffmann-La Roche Limited
Paul E. Prendergast ..... AL 3-1962
Wilf Baldwin ..... HU 9-8236

Horner, Frank W. Limited
Jos. Lavitt ..... JU 9-1691
Earl Ayre ..... CE 3-4086
Frank Lowe ..... VE 2-5821

Lederle Laboratories
J. G. Jonasson ..... SP 5-4862
W. C. Hall ..... HU 9-2207
J. E. Smith ..... AL 3-0437

Merck, Sharp and Dohme (Canada) Ltd.
W. G. Ball ..... GR 5-5702
Noel J. Pritchard ..... GL 2-6880
E. J. Strimbicki ..... AL 3-5767

Merrell Company, The Wm. S.
Norman Haldane ..... VE 2-3909
L. Raymond Scott ..... SP 4-8079

Parke Davis & Co.
L. W. Curry ..... HU 9-1138
B. S. Fleury ..... GL 3-7071
R. J. Robinson (Brandon) 92-288
J. B. Peters ..... VE 2-3862

Pfizer Canada
E. E. Conway ..... TU 8-1193
W. R. Mitchell ..... GL 3-6226
W. G. Johnston ..... TU 8-1391
Blake Johns ..... SP 5-1404

Riker Pharmaceutical Co. Ltd.
John R. Falconer ..... GL 3-5385

Robins (Canada) Ltd., A. H.
Harold Tetlock ..... SP 5-5624
Fred Gallinger ..... ED 4-1367

Sandoz Pharmaceuticals Ltd.
H. D. Robins ..... ED 8-8216

Schering Corp. Ltd.
Halsey Park ..... HU 9-4346
John D. Nicolson ..... LE 3-4447
Allan L. Segal ..... HU 9-7701

Schmid (Canada) Ltd., Julius
H. V. Walker ..... LE 3-8664

Searle & Co., G. D.
Harry Chambers ..... LE 3-6558

Squibb & Sons, E. R.
A. B. Shaen ..... GL 2-9859
M. G. Waddell ..... GR 5-1552

Warner-Chilcott Labs.
A. L. (Andy) Argue ..... TU 8-1619
Gordon Harrower ..... AL 3-4147

Will, Charles R.
A. C. Payne ..... VE 2-2055

Winthrop Laboratories
R. M. Kelly ..... Res. GR 5-6038
A. E. Pauwels ..... CE 3-2024

Wyeth & Brother, John
A. W. Cumming ..... HU 9-5694
Stuart Holmes ..... JU 9-4273